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CONTENTS

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"MY DOCTOR'S MADE A NEW MAN OUTTA BOTH OF US!"

"ALL that endless figuring and re-figuring of milk, carbohydrates, water for feeding formulas was getting my doctor down. 'Specially with all he has to do these days.

"No wonder he looked into S-M-A. An' no wonder he made all his babies S-M-A babies—right off! It sure fixed him up with extra time for his extra work—and even a bit for some sleep. Why, it takes only two minutes to explain to a mother or nurse how to mix and feed S-M-A*.

"*Better yet, my doctor knows that in S-M-A he's prescribing an infant food that closely resembles breast milk in digestibility and nutritional completeness!*

"Happy am I—and so is Mummy! 'Cause S-M-A made a new man outta me. I'm gaining by leaps and bounds. And Doctor? His new disposition matches *mine*. Believe you me, EVERYBODY'S happy if it's an S-M-A baby!" A nutritional product of the S. M. A. Corporation, Division WYETH Incorporated.

**One S-M-A measuring cup powder to one ounce water.*

S-M-A is derived from tuberculin-tested cows' milk, the fat of which is replaced by animal and vegetable fats, including biologically tested cod liver oil, with milk sugar and potassium chloride added, altogether forming an antirachitic food. When diluted according to directions, S-M-A is essentially similar to human milk in percentages of protein, fat, carbohydrate, ash, in chemical constants of fat and physical properties.

"Everybody's Happy

. . . IF IT'S AN SMA BABY!"

REG. U. S. PAT. OFF.

PEPTIC ULCER

PHILIP J. CUNNANE, M.D., F.A.C.S.
Los Angeles, Calif.

THE problem of treatment in peptic ulcer has finally reached a background of unanimity of approach. After decades of disagreement, the Internist and the Surgeon seem now to be in almost complete accord. As that accord has become increasingly evident, the indications for the surgical treatment of peptic ulcer have become even more restricted.

Ulcer of the stomach and duodenum are essentially identical pathologically. The stomach and the first part of the duodenum have the same embryonic origin and blood supply. They are both continuously subjected to acid secretion. They are both prone to recur after response to adequate treatment. Both occur in non-acid producing mucosa in close proximity to acid producing mucous membrane. Acid is the important factor in the producion of each lesion. Because of this similarity of background, ulcer of the stomach and duodenum are considered together as peptic ulcer.

The Medical Military experience of the English Canadians in the present conflict suggests that, next to flat feet, peptic ulcer is the most frequent cause of disability among enlisted men[1]. The comparative incidence in the United States forces has not been determined. It has been found that 90 per cent of the soldiers with peptic ulcer in the Army of the United States had ulcers prior to entering the service[2]. When the symptoms of this group prevent them from carrying on their duties, they are discharged from the service because medical treatment has not been of sufficient benefit and their symptoms are not of a degree that warrant surgery.

The treatment of the uncomplicated peptic ulcer is primarily a medical problem. Well defined complications do develop in a minority of cases that require surgical treatment. The percentages of such complications developing among all peptic ulcers cannot at this time be determined. Our statistics are not adequately encompassing and do not include great numbers of ambulatory patients that are not hospitalized. The majority of patients that are hospitalized for the treatment of peptic ulcer present complications that are generally recognized as potentially surgical. We can state only that approximately 20 per cent of our hospitalized cases are surgical and that more than half of these are treated surgically for perforation.

It has been the effort of the Surgeon over the period of years to establish both an acceptable operative procedure in ulcer and definite indications for the employment of that procedure. These indications may be outlined as follows:

1. Perforation
2. Hemorrhage
3. Obstruction
4. Failure of response to adequate medical treatment
5. Gastrojejunal ulcer and its complications.

Perforation: More than one-third of patients upon our services that have been treated surgically for perforation have presented spontaneous closure of the perforation by implantation of a contiguous structure such as the omentum. This, however, cannot be predetermined and all must be considered as surgical. Perforated duodenal ulcers are practically always limited to the first portion and are usually on the anterior surface. Acute perforations of the stomach occur usually on the anterior wall near the incisura.

In most cases, the symptoms are typical and the diagnosis is obvious. In differential diagnosis, two points may be here considered. Increased serum amylase and diastasuria cannot always be interpreted as indicative of a primary, non surgical acute pancreatitis. The perforated duodenal ulcer may be associated with a degree of duodenitis that results in ampullar inadequacy and a consequent regurgitation into the pancreatic duct. Evidences of the secondary oedematous pancreatitis are reflected in the increased serum amylase and urinary diastase. Secondly, our ability to demonstrate pneumoperitoneum by X-ray in perforated ulcer is increased by 15 per cent with the patient in the left lateral decubitus position.

Read beforre Arizona State Medical Society, Phoenix, April 15, 1944.

The minimal technical effort consists in closure of the perforation and that only should be attempted. The oedematous obstruction at the pylorus will subside under proper postoperative care which includes continuous decompression. In the unusual case, resective surgery may be indicated. We have felt that it was indicated on only two occasions. In each case, the patient perforated in his hospital bed during the period of preoperative preparation for the elective surgical relief of cicatricial pyloric stenosis.

Drainage may be suggested as a basis of controversy. In the hyperacidity of peptic ulcer, the possibility of infection is not formidable. Cultural results in the presence of 30 or more units of free acid in the empty stomach are usually negative. In the massive perforation following a full meal, with extensive and gross peritoneal contamination, drainage may be indicated. The drain should be remotely placed from the line of suture closing the perforation.

Subacute and chronic perforations occur more frequently on the posterior wall of the stomach and duodenum. They are readily occluded or walled off in the lesser omental cavity and become surgical because of a failure to respond to medical treatment.

Hemorrhage may be an indication for surgery and is an alarming complication. Seventy per cent of hematemesis occurs from an intrinsic benign lesion of the stomach or duodenum and approximately 50 per cent from duodenal ulcer alone. Hemorrhage in varying degrees occurs as a complication of peptic ulcer in 20 per cent of all cases.

The chronic minimal type of hemorrhage is evidenced by tarry stools over a long period of time with progressive secondary anemia and its associated symptoms. The more acute, massive type of hemorrhage is manifested by hematemesis and melena, accompanied by syncope, fainting, air hunger, fall in blood pressure, rapid and thready pulse, collapse and the other symptoms of shock. In the slow hemorrhage, adjustment can be made to the diminishing blood volume. The precipitous hemorrhage does not allow for adjustment to the rapid decrease in such blood volume.

Approximately three-fourths of massive hemorrhages occur in patients past 45 years of age and more than 90 per cent of the deaths occur in patients about that age. Age, sclerotic arteries, and the male sex increase both the incidence and the death rate. There is a tremendous increase in the mortality rate following the first hemorrhage in this older age group. Although the more frequent cause of hemorrhage is the duodenal ulcer, the mortality rate is higher in the massive bleeding from gastric ulcer.

Because of the fact that practically the whole mortality rate in hemorrhage from peptic ulcer occurs in the patient over 45 years of age, a hemorrhage after that period of life presents a definite indication for resective surgery following control and adequate preparation. Repeated hemorrhages in the younger individual who has been under proper and persistent medical treatment present the same indication.

The problem of whether or not the currently and grossly bleeding peptic ulcer should be treated surgically has not yet been decided. Our mortality rate in patients so treated does not improve the general mortality rate among similar cases under medical management. Our indications for such interference have been described by Holman: [3, 4]

1. Recurrent or persistent bleeding during or immediately following complete bed rest.
2. Hemorrhage in the hospitalized patient who is under adequate medical treatment.

Muelengracht[5] has stated that he considered a mortality rate of 1 to 2 per cent on dietary medical management makes the question unreal to him and relieves him of the responsibility of having to decide in an individual case whether operation is advisable or not. Thorstad of Detroit, in a more comprehensive review of the problem of the bleeding peptic ulcer, concludes that there is no proof, as yet, that those who attempt the surgical arrest of acute massive hemorrhage from a peptic ulcer are able to do so with a reasonable mortality.

Obstruction of any degree that persists and prevents the patient from following his usual occupation and results in invalidism is an indication for surgery. The criterion of a degree of obstruction that is surgical is not measurable in percentages of demonstrable retention. A minimal obstruction may incapacitate one patient while a greater degree will not interfere with the ability of another patient to carry on with his normal activities. The factual criterion

of a surgical obstruction is progressive vomiting with loss of weight and finally evidences of alkalosis and gastric tetany.

Obstruction with retention may result from one or all of three factors.

1. Spasm
2. Inflammatory oedema
3. Cicatricial stenosis.

Even though the obsruction at the pylorus is complete medical treatment should first be attempted. Inflammatory oedema and spasm as a background of obstruction usually respond to this type of therapy. Cicatricial narrowing does not respond and the degree of such constriction can be determined fully after the spasm and oedema have been made to subside.

This treatment, in addition to overcoming the alkalosis and malnutrition and determining the actual degree of cicatricial narrowing, also by decompression diminishes the size of the distended stomach. Distention more definitely involves the lower two-thirds of the stomach rather than the upper one-third. Measurement of the lower two-thirds of the undilated stomach for resection is not mathematical. In the presence of this eccentric distention, measurement is even more difficult. In the past, we have concluded, on the basis of subsequent X-ray study and the evaluation of acid levels, that we have removed less of the stomach than had been intended because of this mathematical inaccuracy exaggerated by distention. Contraction of the stomach to a more normal gastric volume also results in less distortion of the anastamotic line if surgery becomes necessary.

Medical treatment consists in keeping the stomach empty by suction on an inlying Levine tube, the adequate administration of glucose and saline solutions parenterally, sedatives and anti-spasmodics, and sufficient vitamin intake.

Perpetuation of disabling symptoms under adequate and long continued medical treatment usually presents a positive indication for surgery. A larger and more controversial group falls under this classification. This controversial group contains those individuals whose ulcers are intractable because of a failure to submit to proper medical treatment. The Internist is confronted with multiple evident or expressed reasons that contribute towards this failure. The background may be occupational with an inability not only to follow a consistent medical regime but also an inability to change the type of occupation for economic reasons. Today, defense workers on swing and graveyard shifts have difficulty in regulating their habits of living and eating in conformity with an adequately regulated ulcer regime. Ignorance and apathy may be factors. Alcoholism is often contributory.

We must consider here the problem of gastric ulcer. We cannot predict malignant degeneration and the diagnostic error in differentiating the benign from the malignant lesion will approximate 10 per cent. Since the final diagnosis is made too frequently upon microscopical examination of the resected specimen, our indications for resective surgery must be expanded. Since conservative treatment of the gastric lesion places such high responsibility upon both the Internist and the Surgeon, it must be assumed that the uncomplicated gastric ulcer is always potentially a surgical problem.

The malignant lesion may respond to treatment by disappearance of subjective symptoms, by disappearance of the deformity on X-Ray and of blood in the stool. The lesion whose history is long standing is usually benign. The larger lesion is more frequently malignant. The malignant lesion occurs usually in the older patient. Achlorhydria is characteristic of gastric carcinoma but acid may be present in malignancy. Although all of these findings are corroborative, they are not positive in an attempt at differentiation.

The practise on our services include these positive indications.

1. The gastric lesion located on the greater curvature or within one inch of the pylorus is not at any time a medical problem.
2. The gastric ulcer, whatever its location, that fails to respond to medical treatment both clinically and by X-Ray within four weeks should be surgically treated.
3. That gastric lesion, whatever its location, in which the evidence of history, acidity, the X-Ray and the gastroscope does not strongly suggest a benign character, becomes immediatetly a surgical problem.

The gastrojejunal ulcer and its complications are positive indications for surgery. The principal complications are hemorrhage and perforation. Perforation may occur into the general peritoneal cavity, posteriorly into the

head of the pancreas or lesser omental cavity, or into the transverse colon resulting in gastrojejuno colic fistula. Gastrojejuno colic fistula is a devastating complication. Because of the vomiting of fecal material and the concomitant diarrhoea, the diagnosis is usually obvious. If the fistulous tract cannot be demonstrated by X-Ray upon ingestion of barium, the regurgitation from the colon into the stomach can invariably be demonstrated by barium enema. Preliminary right colostomy has greatly reduced the hazard in the surgical treatment of this lesion. The procedure terminates regurgitation from the colon into the stomach and frequently the fistulous tract into the colon spontaneously closes.

Surgical Treatment: The effort of the surgeon over the period of years has been to establish definite principles of an acceptable operation for peptic ulcer. The attempt during all of this time has been to eliminate the current ulcer and prevent the recurrent ulcer. Confusion existed because we did not know how the peptic ulcer originated. We knew only that it was associated almost invariably with a concomitant hyperacidity. The correlation of studies on the physiology of gastric secretions and gastric mobility particularly as it is related to ulcer, and their application to the development of the criteria for a successful operation have, altogether, resulted in our present accepted procedure.

For the Clinician, a review of the physiology of gastric secretion by Schiffrin and Ivy[6] seems the most acceptable. We feel that Wangensteen[7] has contributed more than any other surgeon in the application of these studies to the surgical treatment of ulcer. He is probably frequently quoted without credit because of an inability to refer directly to his many constructive contributions.

It is not known now definitely what factors change the resistance of the gastric and duodenal mucous membranes and make them susceptible to acid digestion. The factors involved in the change of the capacity of the gastric secretory mechanism to secrete acid are not yet recognized. It is recognized, however, that acid is the important factor in the genesis of ulcer. Ulcer itself is not the disease but in the terminal effect of the action of a highly acid gastric secretion upon the mucous membrane of the stomach and duodenum. This whole background, the mucous membrane with its lowered resistance and the gastric secretory mechanism with its changed capacity, constitutes the ulcer diathesis.

The satisfactory operation then, as epitomized by Wangensteen, is the one that (1) eliminates the diathesis and relieves the patient subjectively, (2) prevents the recurrence of ulcer, and (3) does not compromise the future of the patient. Clinically, it is becoming more generally and increasingly evident that the only procedure that conforms to these requirements is the removal of the lower two-thirds of the stomach. That procedure removes the most usual area in which ulcer originates. It removes that portion of the gastric mucosa that secretes histamine and possibly other substances that activate the secretory function of the acid glands of the fundus. It also removes a part of these fundic acid glands. Because of these accomplishments, the final acid content of the stomach approaches achlorhydria. This depression of the acid content and the removal of the non acid secreting portion of the stomach which is potentially ulcer bearing eliminates the ulcer diathesis, relieves the patient subjectively, and, insofar as we can evaluate our end results at this time, prevents recurrence of the ulcer. Although not so isolated, that intrinsic substance that prevents pernicious anemia must in some part be secreted in the fundus since no deficiency disorder results. The remnant of stomach soon allows of meals of normal amount and regularity.

Gastro-enterostomy alone is never the procedure of choice although occasionally it may be the procedure of better judgment. This occasion may occur when resection is so complicated by inflammatory involvement of contiguous structures in the obstructed patient who is a poor risk that the anticipated mortality rate is inordinately high. In the case not so complicated, the difference in mortality rate between gastro-enterostomy and resection is negligible.

When it is suggested now that the surgical treatment for peptic ulcer is resection of two-thirds of the stomach, it should not be concluded that this statement is incontrovertible. That is the treatment on the basis of our present knowledge and until adverse statistics are accumulaed. With the advent of accumulated

statistics, it may be decided that this procedure is unsound. In the past, gastro-enterostomy was defeated and practiced for a number of years despite the increasing incidence of gastrojejunal ulcers following its practice. We do know now that the incidence of marginal ulcer approximated 25 per cent and with earlier recognition, the practise should have been terminated as an unsound procedure. With such inexperience in our background, resection for ulcer will receive a more meticulous and an earlier evaluation.

BIBLIOGRAPHY

1. O. H. Wangensteen: Surgical Management of Peptic Ulcer a Chemical Problem. Minnesota Medicine, 26:206, 1943.
2. Waltman Walters: Gastric and Duodenal Ulcer Among Naval Personnel. Surg. Gyn. Obst., 78:335, 1944.
2. Holman, C. W.: Severe Hemorrhage in Gastric and Duodenal Ulcer. Arch. Surg., 40:150, 1940.
4. Holman, C. W.: Treatment of Bleeding Peptic Ulcers, Internat. Clin. 3:164, 1940.
5. Muelengracht, E.: Medical Treatment of Peytic Ulcer and its Complications, Brit. M. J., 2:321, 1939.
6. M. J. Schiffrin, Ph.D. and A. C. Ivy, M.D., Ph.O.: Physiology of Gastric Secretion, particularly as relateh to the Ulcer Problem. Arch. Surgery, 44:389, 1942.
8. John W. Cline, M.D.: Gastric Resection for Peptic Ulcer. Calif. and West. Med. 59:7, 1943.
9. M. Jordan Thorstad, M.D.: Problem of the Bleeding Ulcer. Surgery, 12:964. 1942.
10. Frank H. Lahey, M.D.: Surg Practise of the Lahye Clinic. W. B. Saunders, 1942.

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X-RAY FINDINGS IN SILICOSIS

Supplementary Report of the Subcommittee on Silicosis of the Committee on Industrial Health, Arizona State Medical Association

A LARGE part of the work of the Subcommittee on Silicosis of the Arizona Medical Association was given over to a careful study of the literature of the past ten years on the important subject of industrial dust diseases. Individual members of the subcommittee were asked to review and summarize the observations on designated phases of silicosis especially. The history and etiology were reviewed by Dr. John W. Flinn of Prescott; the diagnosis and differential diagnosis were reviewed by Dr. Fred G. Holmes of Phoenix; the clinical course was reviewed by Dr. Louis Baldwin of Phoenix; the treatment was reviewed by Dr. William M. Schultz of Tucson; compensation legislation was reviewed by Dr. Frank T. Hogeland of Cananea, and so forth. An important assignment which was not committed to writing was the one on pathology and x-ray findings. It is probably fortunate that this was deferred, because we now have an opportunity to consider some important points in the pathological development and in the x-ray interpretation, and to harmonize these with the Arizona Occupational Disease Law, which was passed at the 1943 session of the legislature and is now being made effective.

The Arizona law above referred to sets up a definition of silicosis to which radiologists will need to conform their interpretation of x-ray findings, so that the officials charged with the administration of the law can translate these interpretations into the terms of this definition. Item 35, of Section 36, of the Occupational Disease Law, defines silicosis in these words:

"For the purpose of this act 'silicosis' or 'asbestosis' are defined as chronic diseases of the lungs caused by the prolonged inhalation of silicon dioxide dust (SiO_2) or asbestos dust, respecively, characterized by small discrete nodules of fibrous tissue similarly disseminated throughout both lungs, causing a characteristic x-ray pattern, and by variable clinical manifestations."

This definition of "silicosis" was taken from the Utah law, but was unfortunately confused by the introduction of "asbestosis," and the application of the definition to the latter condition, which is not pathologically or roentgenologically correct. Asbestosis does not result in discrete nodular fibrosis and produces a very different x-ray shadow from that of silicosis. The early reaction to asbestos dust, according to Gardner, is diffuse and the x-ray appearance is a generalized diffuse haziness in the lower lung fields; later the appearance becomes finely granular, producing the well-recognized "ground glass appearance." The industrial hazard from asbestosis in Arizona is very slight, at the present time at least, and until this error in the definition can be corrected by the appropriate legislative amendment, the inclusion in it of asbestosis can best be ignored.

The application of this definition, so far as silicosis is concerned, and the classification of silicosis into its progressive stages of development has recently been elaborated by Dr. Leroy Gardner, of Saranac Lake, N. Y., by answers to questions propounded to him in a hearing before the Utah Industrial Commission. Dr.

Gardner outlined the Saranac Lake classification and described the x-ray appearances found in each stage. There are five stages thus described. Two of the stages are prenodular and show only fibrotic striation which is not pathognomonic of silicosis, since it may result from any condition causing peribronchial fibrosis. These two stages he calls P.1 and P.2, the difference between them depending on the degree of fibrotic striation. Since exactly similar changes can, and frequently do, occur in a variety of other conditions, the radiologist cannot make a positive interpretation of silicosis in these stages, and the definition in the Utah law does not recognize these stages as compensable silicosis. Only after nodulation develops can the interpretation of silicosis be made, and these nodular shadows are classified into stage 1 (S.1), stage 2 (S.2) and stage 3 (S.3) silicosis, depending on the number and sizes of the nodular shadows.

Confusion has arisen in Arizona, because some of us have been following the roentgenological classification of Lanza and Childs (see U.S.P.H.S.. Bull. No. 85, 1917), this having become well established as authoritative until recent years. The "first stage" silicosis of Lanza and Childs corresponds to the P.1 and P.2 prenodular stage of Gardner, which he states cannot be definitely interpreted as silicosis and which the Arizona Occupational Disease Law definition does not recognize as silicosis, since nodules have not yet appeared. Lanza now undoubtedly agrees with this, because in his book on "Silicosis and Asbestosis" (1938), the chapter on "Roentgen Ray Diagnosis" written by Pendergrass, discusses Lanza's three stages and has the following comment on the first stage of this classification:

"The general manifestations of this first stage are not characteristic of pneumoconiosis alone, but may be simulated by many other conditions, especially passive congestion, acute or chronic respiratory infections, chronic bronchial catarrh and bronchiectasis. Moreover the hilum and trunk shadows are subject to considerable normal variations in appearance in the adult. As a numerical stage, this one implies, theoretically at least, an early period in progression, yet individuals may remain in this stage for years, or indefinitely. As the manifestations of a typical first stage, with prominent hilum and trunk shadows and linear markings, are not characteristic of pneumoconiosis alone, I am inclined to doubt the fairness of giving this stage a definite medicolegal status, or of using it as a basis for compensation."

In the further elaboration of the subject, Pendergrass summarizes the investigations carried on over many years by Pancoast and himself, and presents their classification, based on a correlation of the pathological and x-ray findings. Pendergrass agrees entirely with Gardner, and we may assume that Lanza is also in agreement. Pendergrass describes "Healthy Lungs and Adnexa" and includes under that heading the P.1 and P.2 fibrotic striation of Gardner's classification, because they are not disabling. He (Pendergrass) says, "Silicosis as a clinical disease begins only when the lung proper is affected", and the x-ray evidence of this is the nodulation which is visible in the roentgenogram of the chest.

With the authority of Pancoast and Pendergrass, Gardner and his associates, and Lanza, back of the Arizona Occupational Disease Law definition of silicosis, we should have no hesitancy in framing our x-ray reports to conform to this now generally accepted classification. Under this definition, we can make an interpretation (diagnosis) of silicosis only after nodulation has appeared and can be distinguished as such on the roentgencgram, and when this nodulation is "similarly disseminated throughout both lungs in a characteristic x-ray pattern," and this in a person with a history of prolonged exposure to silica dust. We should either not use Lanza and Childs' old classification at all (since the Arizona law does not recognize it), or if we do use the term "first stage" as referable to the prenodular linear fibrosis and increased hilar shadow, we should qualify and explain that we are using the term "first stage" in this sense.

It will, however, be less confusing for all the radiologists of Arizona to agree that we will follow the Saranac Lake classification of Gardner, since it now has the weight of all accepted authorities back of it and since it is in conformity with the Arizona law and definition of silicosis. Under this classification, visible nodulation marking the advent of silicosis as a clinical disease, is divided into three stages, called by Gardner S.1, S.2 and S.3. Since the degree of nodulation which marks a silicosis as stage 1, 2 or 3 is not sharply defined, there undoubtedly will be individual variation in

interpretation. However, this is not important since the determination of disability from uncomplicated silicosis does not depend on the amount of nodulation but upon other clinical symptoms which the radiologist is not called upon to evaluate. In several conditions the interpretation of the radioligist is extremely important, aside from the classification of recognizable silicosis.

(1) A differential interpretation is frequently required. There are a surprising number of non-silicotic individuals whose roentgenograms show discrete nodular densities, varying in number from a few to many hundreds, and distributed over the lung fields in a manner quite similar to silicosis. Some of these are the discrete multiple calcifications usually regarded as being the result of healed disseminated tuberculosis of infancy or childhood; these usually are discovered in people who have no symptoms, and many have been uncovered by the Selective Service examinations, school and industrial surveys of large groups of apparently healthy people. When such densities are discovered in a miner or other worker in industrial dust, an important problem is presented to the roentgenologist. The nodular densities of some types of fungus infection in the lungs may be quite confusing, and probably more nearly resemble silicosis than does any other non-silicotic condition. There have been reports from observers of miners in England of fungus infection in the lungs producing shadows indistinguishable from those of silicosis. The mouldy hay fungus, whose lesions in the lungs have been studied by the U. S. Public Health Service, is another example of such confusing condition, which roentgenologists must bear in mind. The densities of miliary tuberculosis can be confusing, especially if first discovered after a period of dust exposure long enough to make silicosis possible. The development of new types of irritant fumes and dusts in the expanding industry of the state will undoubtedly bring us new problems in the interpretation of lung shadows, all of which will demand constant vigilance on the part of roentgenologists, as well as expert knowledge in the interpretation of lung densities and the differentiation of them from silicosis.

(2) Where there is manifest tuberculosis in a worker who claims to have silicosis, the question will need to be determined by the roentgenologist whether there is sufficient nodulation to warrant an x-ray interpretation of stage 1 silicosis. The roentgenologist should answer this question on the basis of the definition set forth in our law and determine whether there are "small discrete nodules of fibrous tissue similarly disseminated throughout both lungs, causing a characteristic x-ray pattern." If the x-ray appearances do not conform to this description, the roentgenologist should not make an interpretation of silicosis. The compensability of the condition does not rest on the tuberculosis, but depends on whether there is an associated *recognizable* and *characteristic* silicosis. An occupational history of long exposure to silica dust, increased hilar shadows and linear striation which *could* be the result of silicosis, and the presence of an active tuberculosis, are not sufficient to warrant an interpretation of tuberculo-silicosis under our law. To make such an interpretation there must first be silicotic nodulation which is recognizable as such on the x-ray film. Lacking this, the roentgenologist cannot make the interpretation and the clinician cannot make this diagnosis. So, there will be the temptation in some cases, to stretch the imagination a little in an effort to satisfy our minds that the nodules are present; or, on the other hand, to assign some other explanation to densities which another roentgenologist might call nodulation of silicosis.

(3) Where there is recognizable silicotic nodulation, with a consistent history, and densities of what seems to be an old tuberculosis, a somewhat different and perhaps more difficult question will be presented. If there is manifest tuberculosis, with cavity formation or other characteristics of activity, or where the sputum is positive for tubercle bacilli, no problem is presented. But if there are apical densities of a pleural cap, or fibrotic changes usually interpreted as healed tuberculosis, or even hilum calcification to the degree which now calls for rejection for military service on the basis of a primary healed tuberculosis, and with these evidences of inactive or obsolete tuberculosis there is also a second or third stage, or even an undoubted first stage silicosis, what is to be our interpretation? Since the interpretation of tuberculo-silicosis can be made only when *both* conditions are active and pro-

gressive, and since the primary healed lesion of childhood and other healed lesions of obsolete tuberculosis are not active, their mere presence along with silicosis does not warrant the interpretation of tuberculo-silicosis. It would seem that the *diagnosis* of tuberculo-silicosis cannot be made from the x-ray interpretation alone and demands the consideration of other factors; hence the final conclusion should be left to the clinician, and the roentgenologist should interpret the two types of shadows as separate entities, without trying to link them into a final diagnosis. In other words, the roengenologist makes an x-ray inerpretation of shadows, but does not make a diagnosis.

To illustrate the changes which develop in the lungs from the inhalation of silica, there are being filed with this report copies of eight films, exhibited by Dr. F. T. Hogeland in the Silicosis Exhibit of this Subcommittee at Prescott in 1942. These are all on the same individual, taken at yearly intervals over a period of eight years, except for one interval of two years. They show the appearances in the lungs of a healthy young man, not previously exposed to silica dust except as he would encounter it while working as a cowboy. He started to work in the mine at Cananea in 1934, and just prior to his employment the first film was made. That film and the subsequent ones taken in 1935, 1936 and 1937 would be regarded as showing no more than the P.1 linear striation, which in his man was evidently due to some old chest infection. In the film of 1938 it might be classed as P.2; in the light of *subsequent* developments, some of this added linear striation and accentuated hilar density is undoubtedly due to beginning silicosis, but the interpretation cannot be made from the x-ray film at this time. The film of 1939 shows nodular densities which warrant interpretation of silicosis, stage 1 (S.1). Two years elapse before the next film was taken in 1941, and here we have definite stage 2 silicosis (S.2). The final film shows further increase in shadow, and the densities presented would be regarded by most roentgenologists as late stage 2 or stage 3 silicosis (S.2 or S.3).

These films present the x-ray densities which represent the tissue changes of a developing silicosis, as will be found described by Gardner and others. The densities shown correspond to the description of Gardner before the Utah Industrial Commission.

W. WARNER WATKINS, Chairman,
Subcommittee on Silicosis.

(Note: The above report was unanimously approved by the newly organized Section on Radiology and Pathology, of the Arizona Medical Association, in Phoenix, on April 14, 1944. Members of the Section present were: Dr. Thomas Hartgraves (Phoenix), Chairman; Dr. H. S. Faris (Tucson), Vice-chairman; Dr. Maitland S. Dirks (Phoenix), Secretary; Dr. E. M. Hayden (Tucson), Dr. R. Lee Foster (Phoenix), Dr. George O. Hartman (Tucson), Dr. A. L. Lindberg (Tucson), Dr. Maurice Rosenthal (Phoenix), and Dr. W. Warner Watkins (Phoenix). Dr. H. P. Mills and Dr. T. R. Moran, both of Phoenix, were absent, but concur in this report.)

VITAL CAPACITY IN SILICOSIS

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INTRODUCTION

BLAISDELL has recently demonstrated that it is possible to estimate disability in silicosis by performing various tests introduced by McCann of the University of Rochester, New York. The tests include X-ray to obtain radiographic volume of lungs, spirometer studies, ergometer records and gas analysis. These tests are chiefly used in estimating the "residual air" which is that air still left in the lungs after total expiration and represents about 22 to 27 per cent of the "total lung volume."

The vital capacity is easily obtained by simple spirometer studies and represents about 75 per cent of the "total lung volume." In preemployment examinations the vital capacity test used alone is of value as an indicator of pulmonary reserve and is helpful in evaluating the disability in silicosis without recourse to the more accurate and time consuming methods employing ergometer records, gas analysis and measurement of radiographic volume of the lungs.

In this study the vital capacity test was employed to estimate the pulmonary reserve in 425 miners. In each case X-ray and physical examination was correlated with vital capacity findings. It is the purpose of this paper to review the various factors affecting vital capacity and to present a statistical study of vital ca-

pacity and incidence of silicosis in an unselected group of 425 miners.

VITAL CAPACITY DEFINED AND CLASSIFIED

Vital capacity is the total amount of air which can be forcibly exhaled after a maximal inspiration. It is best measured by a carefully calibrated and counterbalanced water spirometer. Many examination forms require the measurement of the chest expansion; however, this is at best a poor indication of lung capacity due to the fact that many muscular individuals can enlarge the thorax to a greater degree than the lungs are capable of expanding.

First, Hutchinson[7] and, later Dryer[5] and West[10] found that the vital capacity bears a consistent relationship to the surface area of the body. A less accurate relationship was also found between the height and the vital capacity. The difference in the results of these two methods of calculation is about 5 per cent.

Vital Capacity, c.c. per cm. height—

Men	Women	Athletes
25 c.c.	20.0 c.c.	29.0 c.c.

Vital Capacity, c.c. per square meter—

Men	Women	Athletes
2500 c.c.	2000 c.c.	800 c.c.

Dryer[5] and other investigators found that the occupational activity influenced vital capacity. Sedentary workers were found to have a much lower vital capacity than those engaged in heavy labor. Dryer[6], therefore, established three normal classes—A, B, and C. In class A are those normal individuals with maximum vital capacities, as shown in the above table. Class B (not less than 90 per cent of class A) included those normal individuals who engaged in moderately active physical work. Class C (not less than 85 per cent of class A) included those normal individuals in sedentary occupations. Most miners are considered a reduction of 15 per cent below the standard of the class to which the subject belongs as an almost certain indication of some abnormality.

FACTORS INFLUENCING VITAL CAPACITY

When the vital capacity is calculated on the basis of surface area in square meters, it will be noticed that the obese individual, especially the stocky, thick necked, barrel chested type, is at a decided disadvantage. The vital capacity will be found low in most instances. A two meter X-ray film will show a relatively short, pyramidal shaped lung. This explains in part the rapid development of dyspnea in this type on physical exertion. Add the burden of certain types of silicosis to further reduce pulmonary capacity and dyspnea becomes noticeable even when the individual is at rest.

Tall, long chested individuals if normal physically will usually have the highest vital capacities. Two meter X-ray films will show a relatively large lung as compared with the lung of the short, stocky, obese individual. Tall, thin chested individuals, the lean lanky type, will often have surprisingly high vital capacities. Other physiological conditions in normal individuals which may change the vital capacity measurement is anxiety or nervousness. Apparently a certain tenseness of the respiratory muscles including the diaphragm keeps the individual from deep inspiration and forceful expiration. The examiner should repeat the test at a more favorable time if such appears to be the case. A slight increase in blood pressure or pulse rate in an otherwise normal individual often gives the examiner a hint of underlying anxiety in the subject. Vital capacity measurement is best made both before and after the standard cardiovascular test. It will be found that the vital capacity is often slightly increased after exercise.

Occasionally the examiner will encounter an individual who will not be able to "catch on" as to what is meant by deep inspiration and forceful expiration. They do not seem to understand how to blow and often stop blowing before the breath is spent or start blowing before a complete inspiration is accomplished. A little patience and repetition will usually give an accurate vital capacity in these cases. Some individuals believe that they can increase their vital capacities by a "little practice." Usually, however, the vital capacity measurement is no better, often worse after the tenth or twentieth attempt than after the first few trials.

Vital capacity determinations can be calculated on the basis of ideal weight. This calculation will help determine the approximate correction for the factor of excess obesity or leanness. For example, a low vital capacity in an obese individual recalculated on the basis of ideal weight might raise the percentage to nor-

mal. If the corrected vital capacity is still 15 per cent below the class to which a subject belongs, some abnormality probably exists.

Pathological states which lower the vital capacity are various:

1. Pulmonary disease.
   (a) pneumoconiosis
   (b) tuberculosis
   (c) emphysema
   (d) upper respiratory infection
   (e) asthma
2. Intrinsic mechanical interference with respiratory function.
   (a) pericardial effusions
   (b) pleural effusions
   (c) pneumothorax
   (d) tumors, etc.
3. Intrinsic mechanical interference.
   (a) abdominal or pleuritic pain
   (b) abdominal tumors
   (c) rheumatic or arthritic disease of the costovertebral and sternal joints
4. Heart disease. (Frequently associated with advanced silicosis).
   (a) enlargement of pulmonary vessels
   (b) pulmonary edema
   (c) cardiac dilatation or enlargement

The X-ray film of the chest gives a static-impression of differential capacities in the lung substance and from such an impression the physician often assumes that he can tell the functional respiratory capacity of the applicant. The physician is not only interested in the amount of capacity shown in the X-ray film but more important in the functional loss caused by this opacity. Off hand, one might reason that the more opaque the pulmonary tissue became to the X-rays the greater the functional loss. But this is not invariably true. Great reduction in vital capacity can occur with very little X-ray opacity of pulmonary tissue. On the other hand, the vital capacity is often found to be normal in moderately advanced silicosis.

The peribronchial type of silicosis, described in detail by Pancoast[8] can be so easily confused with chronic passive congestion, bronchiectasis, acute or chronic bronchitis and bronchial irritation due to irritant gases that it was thought best to classify these cases roentgenologically under the heading "accentuated hilar and linear markings". These markings are sometimes so dense in the X-ray film that the examining physician would assume a definite reduction in respiratory capacity in such an individual and further assume that he would be a poor industrial risk. However, vital capacity tests show that respiratory function is very good and the vital capacity is often reduced little or none.

The nodular type of silicosis[8] is characterized roentgenologically by discrete white spots on a darker background of well ventilated lung. These nodules are distinct in the earlier stages of nodular silicosis, but in the later stages the nodules coalesce to form larger masses. The nodules or masses tend to be bilateral, symmetrical, and mostly in the mid-lung fields. The vital capacity in nodular silicosis (frequently but not always) is reduced in proportion to the tendency of increase and coalescence of nodules. Nodular silicosis, affects the highly expansile primary lobule of the lung is effective in reducing vital capacity.

It must be emphasized that various pathological conditions may cause a reduction in vital capacity. For example, obesity and cardiac disease in one individual could seriously reduce the vital capacity. even though the X-ray examination of the lungs shows negligible signs of silicosis. On the other hand, if definite X-ray evidence of silicosis is present along with a low vital capacity one should rule out other possible contributing factors.

ROENTGENOLOGICAL CLASSIFICATION AS USED IN THIS SERIES

I. Chest films characterized by "accentuated hilar and linear markings."

Grade I: Bilateral increase in density confined to the hilus.

Grade II: Bilateral increase in density of the hilus and middle lung fields accompanied by small nodule formation following the pattern of the pulmonary tree.

Grade III: Bilateral increase in density of the hilus and all parts of the pulmonary tree accompanied by nodule formation more pronounced than in Grade II. The degree of accentuation is greater than in Grade II. A "diffuse" increase in density is less frequently noticed.

II. Nodular Silicosis: Characterized roentgenologically by discrete round or oval nodules tending to coalesce, most numerous in the middle lung fields and bearing no definite relationship to the bronchial or vascular tree.

Grade I. Scatttered, but definite nodule formation mostly in the mid-lung field bearing no definite relationship to the bronchial or vascular tree.

Grade II: Numerous discrete nodules throughout both lung fields.

Grade III: Coalescence of nodules to form dense areas typified by the "pawnbroker sign" where the three lobes of the right lung lie adjacent.

STATISTICAL STUDY

Twenty-eight (6.5%) of the 425 miners had nodular silicosis. Of these 11 (40%) had vital capacities below 70% and 17 (60%) had vital capacities over 70%. The incidence of low vital capacity (below 70%) in a group of miners showing no roentgenological evidence of lung or heart disease was 3.5%, as compared with 40% having vital capacity below 70% in the group. The low vital capacity in the normal group was due to obesity, whereas the low vital capacity in the silicotics was due to lung or heart pathology. The apparent high vital capacity (above 70%) in some silicotics was due in several instances to leanness or emaciation. When the vital capacity is calculated on the basis of ideal weight, these underweight silicotics have vital capacities below 70%. In questionable cases, this factor of ideal weight should always be considered.

Eight per cent of the 425 miners examined had enlarged hearts and all but two of this group showed clinical evidence of heart disease (mostly hypertensive vascular disease with cardiac enlargement.) The incidence of heart enlargement was over three times greater in the individuals with advanced (Grade III) silicosis than in the remainder of the group. However, the average age of the group with advanced silicosis was 48 years and the incidence of hypertensive heart disease increases with age. The problem of heart disease as related to silicosis requires further study.

The incidence of the adult form of tuberculosis was 6.1%, and of the 425 cases only 3 or 0.7% were active cases. The incidence of tuberculosis is generally considered high in silicosis, but in this group the incidence was surprisingly low.

Again I wish to emphasize that the recording of vital capacity in pre-employment examinations is not intended to act as a test to determine the amount of disability in cases of silicosis, but only as a test to determine pulmonary reserve and guide the physician in his clinical appraisal of the prospective employee.

SUMMARY

1. Vital capacity determination is a simple, inexpensive and valuable aid in pre-employment examinations to determine the pulmonary reserve of the lungs in those individuals exposed to industrial dust hazards. X-ray examinations of the chest constitutes only partial and incomplete evidence concerning the condition of the respiratory apparatus. Correlation of roentgenological findings with vital capacity determinations gives the examiner added information.

2. Increased linear and hilar markings as seen in the X-ray film of the chest does not seem to be related to serious impairment of the pulmonary reserve. Uncomplicated, early nodular silicosis shows little or no impairment of pulmonary reserve whereas advanced and complicated silicosis definitely causes serious loss of pulmonary reserve.

ther exposed to harmful concentration of silica dusts. An individual having a normal vital capacity below 70% should be considered a poor industrial risk if that individual is to be fur-

3. An individual having definite roentgenological signs of silicosis with a vital capacity should be considered a fair industrial risk despite definite roentgenological evidence of uncomplicated silicosis.

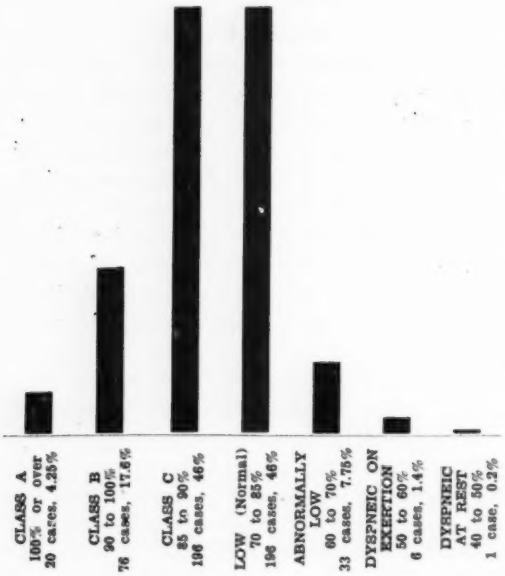
4. Accentuated or increase linear and hilar markings were found in 73% of the miners. Six and five-tenths per cent of the miners had nodular silicosis. Eight per cent of the 425 miners had enlarged hearts, the incidence of en-

CHART SHOWING INCIDENCE OF SILICOSIS

Accentuated Linear Markings	% of Total	Average Vital Capacity	Average Age	Average Yrs. Underground
Grade I	41.8	81 50-106	29.4 18-48	3.6 1 mo-30 yrs
Grade II	27.1	81.5 65-105	32 20-49	6.2 4 mo-25 yrs
Grade III	4.2	75.5 63.9-100	44.5 19-47	13 1 mo-18 yrs
Normal	20.5	80.3 60.5-93	36.5 26-55	1 yr-1 mo 4 yrs-28 yrs
Nodular Silicosis Grade I	.23 4 cases	83.3 (79-86)	25.5 (24-26)	4.5 2 yrs-7 yrs
Grade II	2.7 (12 cases)	71% (79-86)	37 (26-51)	18 (5 mo-37 yrs)
Nodular Silicosis Grade III	2.7 (12 cases)	68.8 40-81	46.8 32-58	14.7 5 yrs-35 yrs

largement being three times greater in those individuals with advanced nodular silicosis. Incidence of pulmonary tuberculosis was 6.1% with 0.7% proven to have active tubercular lesions.

DISTRIBUTION OF VITAL CAPACITY IN 425 MINERS



5. Although miners are moderately active and should have vital capacities between 85% and 90%, this study shows that they have average vital capacities in the low normal group. In other words, the pulmonary reserve of the average miner is definitely below par. On the other hand, only 9.35% of the 425 miners examined had vital capacities below the critical level of 70%.

BIBLIOGRAPHY

1. Arnett, J. H., Vital Capacity of the Lungs; changes occurring health and disease. J. Clin. Inves., 1935, 14,543.
2. DuBois, E. F., Basal Metabolism in Health and Disease. Lea & Febiger, 1937. 6, pp. 119-120.
3. West, C. H. and Taylor, N. B., The Physiological Basics of Medical Practice. 2nd Edition, Williams and Wilkins, 1939. XXX, pp. 505-512.
4. Cole, L. G. and Cole, W. G., Pneumoconiosis (Silicosis). John B. Pierce Foundation, New York, 1940.
5. Dryer, G. Lancet, 1919. 2,227.
6. Dryer, G., The Assessment of Physical Fitness, etc., London. 1920.
7. Hutchinson, J. Todd's Encyclopedia of Anatomy and Physiology. 1850, 4 p. 1016.
8. Pancoast, H. K., and Pendergrass, E. P., Pneumoconiosis (Silicosis). pp. Hoeber, 1926.
9. Pancoast, H. K., and Pendergrass, E. P., Roentgenologic Aspect of Pneumoconiasis and its differential diagnosis. Jour. Am. Med. Assn. 101, 587-591. August 19, 1933.
10. West, H. F. A. Arch. Int. Med., 1920, 25, 306.
11. Blaisdell, JIL, Disability Evaluation in Silicosis. Proceedings of the Third Annual Congress on Industrial Health. Chicago, January 13-14, 1941.

SULFONAMIDES IN THE TREATMENT OF CHRONIC BRONCHIAL INFECTIONS

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INTRODUCTION

THE sulfa drugs have so far been found of greatest value in the treatment of acute infections. They have been found somewhat helpful in certain subacute and chronic infections. Their effect has been least remarkable when the lesions are walled-off or in poor contact with the circulation, or when the causative organism has been poorly affected by the drugs.

The treatment of pulmonary and bronchial infections has been considered to conform to this general pattern. Bronchitis, bronchiectasis, and lung abscess have not been noted to be frequently or dramatically cleared.

Bronchiectasis is one of the conditions which present themselves in Arizona in an advanced but hopeful state. Some improvement may follow residence here, but by their very nature the lesions continue to cause unpleasant local symptoms. The usual prognosis of bronchiectasis is quite gloomy, with a downhill course, marked by exacerbations and complications.

The causes of bronchiectasis are still controversial. Acute disease of the bronchial walls (often virus in origin); chronic sinus infection; bronchial obstruction; distortion of the lung by fibrosis or atelectasis; and even heredity and stress factors may play lone or combined parts in starting or continuing the condition.

The destruction of the mucosa, the fibrosis in the walls, the dilatation and stenosis of the lumina, and the pooling of the purulent secretions are all results which prevent ready healing. The habit-formation, an occasional mental sluggishness, and the very chronicity may also obstruct the use of helpful methods.

TREATMENT METHODS

Clinicians have leaned heavily upon postural drainage as their best single method of treat-

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ment. Bronchoscopy has been helpful in some cases, when available. Removal of a fibrotic and pus-filled lobe from one or even both lungs has been modern and, at times, curative. Unfortunately, many patients are not eligible for surgery, especially if it must be bilateral. Most patients and physicians would avoid surgery if possible.

Sulfonamides have been suggested as treatment for bronchial infections by several authors but the results have been published by very few. Norris reported ten cases in which the drugs had been used, but this was incidental to a study of the concentration of drugs in the bronchial secretions and a study of the effect on respiratory flora. He gave sulfadiazine or thiazole in full doses orally for from four to fifteen days. The concentration in the bronchial secretion was about 60% of the blood level. He noted that in every case there was a marked reduction in the volume of sputum. Daily sputum volumes ranged from 12 to 285 cc. and were reduced from 55% to 81% for an average reduction of 69%. It was difficult to judge the effect on the bronchial flora, since spontaneous changes often occurred, but in general the susceptible organisms were reduced in number (strepococci, H. influenzae, Neisseria catarrhalis, etc.) He recommended the oral use of the drug, preferably during the hospitalization, and the combination with bronchoscopic aspiration in any non-surgical case.

Arizona's John Stacey used the method of Castex to introduce sulfa drugs into the bronchial tree. He described symptomatic improvement in a small series of cases when ½ to 1 cc. of a 5% solution of sodium sulfathiazole was nebulized into the lungs once or several times a day. He mentioned three tuberculous patients with chronic purulent lung infections and two chronic bronchiectatics who were benefitted.

Norris considered that insufflation was too ineffective, however, and that the drug concentration lasted too briefly, especially in purulent secretions.

CASE MATERIAL

The present study is drawn from the use of sulfadiazine, sulfamerazine and occasionally sulfathiazole in 48 cases with purulent bronchial secretion. In some of these the diagnosis was chronic bronchitis (11 cases); in some, bronchial asthma with bronchitis (11 cases); and in some, bronchiectasis (16 cases). There were also 10 cases in which the bronchitis was associated with tuberculosis (6), or strange complications which modified the process (4).

The bronchiectasis cases form the major part of the report. Data on the other cases are mentioned incidentally and for contrast.

Attention was drawn to the value of sulfonamide therapy by experimentation with its use in other chronic infections, and by a chance usage. A patient with severe bronchiectasis was treated with sulfadiazine for a meningococcic meningitis. By the tenth day the meningeal disease was controlled and the sputum had abruptly decreased from 200 to 10 cc.

METHODS OF STUDY

The methods of study were simple. All but three of the patients were ambulatory. They had all been fluoroscoped and X-rayed. Bronchograms had been done at some previous time on all those with bronchiectasis. Sputum quality had been checked by observation; sputum quantity measurement or repeated estimation in common terms. No bacterial studies were done except to exclude tuberculosis. Blood counts were done only as indicated, or at intervals in cases with long drug usage. Fluids were forced, and gross observation of the urine was urged. Alkalinization was not regularly used. Every patient was requested to report by telephone every 48 hours until used to the drug, or whenever any of a list of toxic symptoms occurred.

X-rays or transillumination had been done in each case of sinus disease. The presence of an allergic diathesis was determined chiefly by history; skin-tests were used only where detailed data were desired.

Patients with acute respiratory infections have been excluded from the series. All cases had purulent sputum to some degree.

All patients had lived in Tucson for a control period before chemotherapy was tried. Most of those with bronchiectasis had tried postural drainage abortively in the course of the disease, but were reinstructed in the procedure for a fair trial before the use of sulfonamides.

RESULTS OF TREATMENT

A. An analysis of the series with pure *bronchiectasis* shows the following points of interest:

1. The age of the patients was from 9 to 43 years with an average of 24.5 years.

2. The duration of symptoms varied between 1 and 25 years, with an average of 8.1 years.

3. Fifteen of the 16 cases had a well-established chronic sinus infection.

4. Only two of the cases had gross atelectasis.

5. The daily volume of sputum ranged between 20 and 800 cc, for an average of 173 cc. per patient. It was noted to be predominately purulent in all cases.

6. Sulfadiazine was the sole sulfonamide used in five cases; sulfamerazine was used alone in three cases; diazine and merazine were each used in six cases and these two and thiazole were tried in sequence in three cases.

7. The entire series is too small for evidence on tolerance. It was similar to that reported in the literature. In only one patient was it impossible to find a suitable drug. Diazine and merazine were equally well tolerated except in two cases. Thiazole was the best drug in two cases.

Symptoms of intolerance were the same as usually reported. Hematuria and skin rashes were very rare.

8. The results of treatment of *bronchiectasis* are as follows:

a. The sputum was reduced in all cases. The percent reduction was 95, 80, 80, 95, 50, 50, 50, 80, 80, 60, 50, 30, 25, and 40% in the various cases. The average was a 62% decrease. The least reduction was in the cases having atelectasis, cystic disease, and an odorous sputum.

b. A notable clinical gain was made in 14 of the 16 cases.

c. An improvmeent in the sinus condition was noted in nine cases.

d. Neither of the two cases with recurrent hemoptyses had further bleeding after start of the treatment.

9. Symptoms tended to recur in a period of weeks to months after treatment was discontinued. Acute respiratory infections always resulted in a need for re-starting the drugs.

10. Drug dosage could be reduced to half a gram three times a day (sulfadiazine), with continued freedom from symptoms.

11. A bronchogram was repeated for comparison in only one case, ten months after the start of treatment. There was no remarkable change in bronchial dilatation.

B. The 11 patients with *bronchitis* were slightly different clinically from those with bronchiectasis. Only one had a notable sinus infection; none had atelectasis. The sputum volume averaged about 10 cc., was muco-purrulent in all cases. The drug administration was usually limited to two weeks; the tolerance was average.

Five patients were noted to have made a clinical gain after use of the drug. Surprisingly, the sputum decreased in volume in 7 of the 9 cases, usually being reduced to from 50 to 80%, and often being called less purulent.

C. The 11 patients with *chronic bronchitis and a clinical allergy* showed the following characteristics:

1. Four had sinus infections.

2. All had asthma; five also had allergic rhinitis; four also had hay fever.

3. The sputum volume was slightly greater than in pure bronchitis (25 cc.), but was also muco-purulent in all but one case.

4. The drug tolerance in this allergic group actually was a bit worse than in the other groups. Four patients were made ill by the drugs—about double the usual incidence.

5. Only three of the patients were noted to have gained clinically when the sulfa drugs were in use. Only three of the 11 noted a definite decrease in the sputum volume (50, 30 and 75%).

D. The six patients with *pulmonary tuberculosis and an excessive bronchitis* were not remarkably affected by sulfonamides, probably due to the indefinite source of sputum. Only one was infectious. The drug tolerance was fairly good. Three of these patients were helped immediately on several occasions for acute increases of purulent secretions.

E. Four cases were so complicated as to be considered separately.

(a) A white male, aged 21, had had a purulent bronchitis for two years, metastatic lymphnode abscesses, and a subacute peritonitis. Sulfadiazine repeatedly reduced his sputum and fever, but never produced complete clearance.

(b) A white male, aged 55, was found to have ancient and contracting silicosis, with

hard massive tuberculous lesions near the upper hila. The purulent bronchitis was of two years' duration. The 200 to 400 cc. of sputum decreased 50% in a week, and he became much more comfortable.

(c) A white male, aged 24, had come to Tucson to avoid surgery for a migrating lung abscess. Sulfa drugs helped clear his sputum, but failed to prevent a putrid recurrence.

(d) A white male, aged 48, had developed a sub-diaphragmatic abscess after a gastric resection. This produced a fistula through the diaphragm, pleura, and into the bronchus. A purulent sputum and drainage had existed for months. Both sulfadiazine and merazine produced an almost immediate healing, a 100% decrease in sputum, and an improved general condition.

SUMMARY AND CONCLUSIONS

1. The administration of sulfonamides by mouth has been found regularly effective in all cases of simple, uncomplicated bronchiectasis. Atelectasis and putrid secretions have been associated with a decreased efficiency of the drugs.

2. Tolerance has been about as expected except in cases known to be clinically allergic, where it was decreased.

3. There is no reason to believe that the lesion structure of bronchiectasis will change, though progress may be prevented by sulfonamide treatment.

4. It is recommended that sulfonamides be used in non-surgical cases and for pre-operative therapy. It should be combined with such agents as postural drainage, bronchoscopic aspiration, climate, and treatment of the sinuses.

5. Close clinical control of patients taking sulfa drugs should be exercised.

6. The imminent availability of penicillin for similar cases will make an interesting contrast study.

PEPTOGENIC ULCER IN MECKEL'S DIVERTICULUM

(Case Report)

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MECKEL'S diverticulum is derived from the omphalomesenteric or vitelline duct which in early fetal life connects the mid gut with the yolk sac. Normally, the duct closes during the fifth week of fetal life. A Meckel's diverticulum results when the distal end closes and the ileal end remains open; it is located usually from one to three feet proximal to the ileocecal valve and is almost always on the anti mesenteric border of the ileum. Occasionally, it has a mesentery. This diverticulum occurs in about 2% of all people with a ratio of about 2 males to 1 female.

Histologically, Meckel's diverticulum has the same coats as the ileum and is usually lined with ileal mucosa. In about 25% of diverticula heterotopic mucosal variants are found[1]. The chief variants are gastric mucosa, duodenal, jejunal and pancreatic. Schaetz[2] in a study of 32 specimens found gastric mucosa in 16.6%, duodenal and jejunal 10%, pancreatic 2.7%. Hudson and Koplick[3] in 23 specimens studied found gastric mucosa in 52%. Hudson[4] in 13 cases reported gastric mucosa in 7 and duodenal in 1. Carlson[5] in a study of 152 cases of Meckel's diverticulum found heterotopic tissue in 27.6%. Sacks[6] states that an average figure of 13% represents a conservative estimate of the incidence of gastric mucosa in Meckel's diverticulum.

The chief complications of Meckel's diverticulum are intestinal obstruction, diverticulitis with or without perforation, peptogenic-like ulceration with complication of hemorrhage or perforation or both, intussusception, foreign body and neoplasm.

Denecke[7] was the first (1902) to report an ulcer in Meckel's diverticulum. Deetz[8] five years later pointed out the occurrence of gastric mucosa and stressed the peptogenic character of ulcer in Meckel's diverticulum.

Schaaf[9] in his analysis of the fluid contents of the diverticulum removed by him found it to contain 40% free hydrochloric acid and 70% total acidity. Matthews and

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Dragstedt[10] showed experimentally the small bowel becomes progressively more sensitive to gastric juices as the distance from the stomach increases. They have produced typical chronic ulcers of the Meckel's diverticulum type by anastomosing small Pavlov pouches to the lower end of the ileum. Lindau and Wulff[11] pointed out the secretion of the hydrochloric acid from the gastric mucosa in a Meckel's diverticulum, "begins or increases synchronously with the activity of the stomach —a time during which the small bowel is empty and when no neutralization takes place by food and intestinal juice. In such manner, especially favorable conditions are created for the generation of peptic lesions, a circumstance we find borne out by an occurrence of these ulcers in Meckel's diverticulum even at an early age." It is their belief the process in Meckel's diverticulum supports the biochemical theory of the origin of the ulcer.

Reviews of the literature of peptic ulcer in Meckel's diverticulum were made by Johnston and Renner[12] in 1934 and Cobb[13] in 1936. Cobb reviewed 100 cases of ulcer or probable ulcer. In 66 cases gastric mucosa was demonstrated microscopically. In the remaining cases no microscopic examination was made or no gastric mucosa was found. Seventy-four percent of the cases fell in the age group of 15 years or under, ages varied from 2 weeks to 53 years. Eighty-one of the ulcers occurred in males and 14 in females. In 5 the sex was not stated; the sex distribution approximated that of duodenal ulcer. The principal symptoms were abdominal pain (80%) (51 of 55 perforated cases and 29 or 43 non-perforated cases) and intestinal hemorrhage (72%). Hemorrhage occurred with equal frequency in the perforated and non-perforated groups. Next to hemorrhage perforation was the most frequent and certainly the most serious complication. It occurred in 55 of the 100 cases and there was a mortality of 36%.

The ulcer is usually single but may be multiple. The ulcer has been found, with few exceptions, at the base of the diverticulum at the junction of gastric and ileal tissue rather than within the heterotopic mucosa. In this manner these ulcers are analogous to marginal ulcers following gastroenterostomy. The ulcers may be acute or chronic. They are subject to the same complications as duodenal or gastric ulcer, hemorrhage, perforation and obstruction. The latter may be extrinsic or intrinsic. Waugh, Herrell, and Crumpacker[14] reported two cases of intrinsic obstruction due to scarring of ulcers in Meckel's diverticulum.

The cardinal findings of ulcer in Meckel's diverticulum are hemorrhage, abdominal pain, and incidence in the younger age group. The complication of hemorrhage and perforation occurs most frequently in individuals below the age of 15 years.

Hemorrhage is the most constant symptom. It is usually sudden in onset and massive in character, and it may be so profuse as to be the direct cause of death. The blood passed by rectum may be bright red or black, tarry, unclotted or partially clotted. If perforation does not develop, the patient is likely to recover from hemorrhage and have recurrence at a later date. The importance of hemorrhage and its recurrence over a period of days or months or even years as a diagnostic sign was pointed out in 1932 by Mason and Graham[15]. Hemorrhage may occur simultaneously with perforation or may precede it by days or months.

Abdominal pain may simulate that of duodenal ulcer. Cobb has pointed out the pain may be periodic and colicky; postprandial pains usually occurred in adults. In children particularly, the only history of pain was usually with the occurrence of perforation. With frank perforation the pain is characteristic of perforated viscus with peritonitis.

The diagnosis is based on the age of patient, massive intestinal hemorrhage and abdominal pain. Rousseau and Martin[16] reported a case in which a preoperative diagnosis of Meckel's diverticulum was made by X-ray, and in the survey of the literature found 12 other cases reported. The diagnosis by X-ray is not satisfactory. In the differential diagnosis the exclusion of the common causes of massive intestinal hemorrhage and abdominal pain must be made.

The surgical treatment is resection of the diverticulum with a cuff of the ileum and closure of the defect, transversely, or resection of the diverticulum with a portion of the ileum and repair either by end-to-end or lateral anastomosis.

The mortality in the uncomplicated case is practically nil; however with massive hemorrhage and perforation the mortality is high.

CASE REPORT

A 23-year-old white male was admitted to the hospital on November 17, 1942, complaining of severe pain in the mid abdomen. About 4 a. m. he was awakened from sleep with sudden severe knife-like pain in the region of the umbilicus. At the time of onset of symptoms, he became nauseated, remained nauseated, but did not vomit. He continued to have acute abdominal pain which became progressively severe. At no time did he have bloody or tarry stools. There was no history of previous gastrointestinal complaints.

The past history was not remarkable except for fracture of left humerus in 1933. The family history was not contributory.

Physical examination revealed a well nourished and developed male, 5 ft. 8 in. in height, weighing 140 lbs., and was lying in bed with legs and thighs flexed. He was acutely ill; his cheeks were flushed. He was conscious and co-operative.

Head, eyes, ears, nose, mouth, throat and neck were all negative. The chest was symmetrical; respiration was shallow and of the thoracic type. The lungs were negative to palpation, percussion and auscultation. The heart rate was 108, BP 126/78, otherwise the heart was negative.

Over the lower two-thirds of the abdomen there was board-like rigidity and tenderness; however this was more marked in the region of the umbilicus. In the latter area there was both maximum rebound and referred rebound tenderness. Peristalsis was diminished. Brittain's sign was negative. Rectal examination revealed some tenderness high up on either side, Genitourinary findings were negative, side. Genito-urinary findings were negative, and the extremities were normal.

The laboratory procedures consisted of a blood count which showed RBC 5.01, HB 83%, WBC 15,200 with 84% polys, 3 juveniles, 23 stabs, 58 segments. Urinalysis was negative

A preoperative diagnosis of an acute surgical abdomen, probably due to a perforated ulcer or a perforated appendix, was made.

The abdomen was explored under spinal anesthesia supplemented with ether. When the abdomen was opened, a considerable amount of non-odorous, slightly grayish fluid was found in the lower abdomen, most of which appeared to be on the right side. The peritoneum showed moderate punctate injection, characteristic of that seen in perforated peptic ulcer. The appendix was thick, injected, and gave the appearance of a peri-appendicitis. The terminal ileum was investigated and a Meckel's diverticulum was found about 20 inches proximal to the ileocecal valve. At the junction of the ileum and diverticulum, anteriorly, there was a matchhead size perforation. The diverticulum was roughly cone shaped and measured 4 cm in length and about 2 cm in diameter at the base; it was indurated, edematous, and maximum reaction was around the site of perforation. There was a short thick indurated mesentery. The diverticulum was freed from its mesentery and was resected along with cuff of the ileum. The defect in the ileum was repaired with two rows of continuous catgut suture and reinforced by attaching a free piece of omentum to the line of suture. Five grams of sulfathiazole were sprinkled in the abdominal cavity. A penrose rubber drain was inserted down to the peritoneum at the lower angle of wound.

The microscopic examination of the diverticulum showed on one end of the section villi which were consistent with glands of the ileum. This type of mucosa abruptly changed into a thick layer with columnar type of surface epithelium and elongated glands most of which were in cross sections. The inner-most glands were slightly dilated and contained a parietal type of cell scattered among the larger chief or secretory cells. In one area the mucosa was completely denuded and there was necrosis of the entire wall. This was associated with infiltration of polymorphonuclear leukocytes. The muscularis mucosa was intact except in the area of ulceration. In the musculature there was a fairly diffuse lymphocytic and leukocytic infiltration throughout the section. The serosa was moderately thickened and there was purulent exudate present which was most marked around the area of necrosis. The diagnosis was

Meckel's diverticulum containing gastric mucosa and a perforated ulcer.

The patient was quite ill for the first two postoperative days. He developed a suppurative cellulitis of the operative wound which was incised and drained on the eleventh postoperative day. The patient was last heard from nine months after operation at which time he stated he had continued to be well.

CONCLUSIONS

The cardinal findings of peptogenic ulceration, intestinal hemorrhage, abdominal pain, and incidence in the younger age group; the complications, perforation, hemorrhage, intestinal obstruction in a Meckel's diverticulum were briefly reviewed.

Case report of a young male adult with acute perforation of ulcer in Meckel's diverticulum containing gastric mucosa of the acid secreting type without previous history of abdominal complaints or intestinal hemorrhage was presented.

BIBLIOGRAPHY

1. Matt, J. G., and Timpone, P. J. Peptic Ulcer of Meckel's Diverticulum: Case Report and Reviews of the Literature. Am. J. Surg. 47:612-622, 1940.
2. Schaetz, G. Beitirage zur morphologie des Meckleschen Divertikels, Beitr z. path. Anat. 74:115, 1925.
3. Hudson, H. W., Jr. and Koplick, Harold H. Meckel's Diverticulum in Children: New England Jour. of Med. 206: 827-840, 1932.
4. Hudson, H. W., Jr. Meckel's Diverticulum in Children: 2nd Clinical and Pathology Study with Reports of 13 Additional Cases. New England Jour. of Med. 208:525-535, 1933.
5. Carlson, L. A. quoted by Waugh (14).
6. Sacks, Milton S. Massive Intestinal HeHorrhage from Meckel's Diverticulum: Report of 2 cases. Bulletin of the School of Medicine, University of Maryland 27:197, April, 1943.
7. Denecke: Ueber die Entzuendung des Meckelschcen Divertikels und die Gangraen desselben. Dutsch Ztschr. f. Chir., Vol. 62:523, 1902.
8. Deetz, E.: Perforations peritonitis von einem Darmdivertikel mit Magenschleimhautbau ausgehend. Deusche Ztschr. f. Chir., 88:482, 1907.
9. Schaaf, G.: Beitrag zur Bildung des Ulcus Pepticum in Meckelschen Divertikel Deutsch. Ztschr. f. Chir., 238:78-87, 1932.
10. Matthews, W. D. and Dragstedt, L. R. Etiology of Gastric and Duodenal Ulcer, Especially in he Light of Ulcers in Meckel's Diverticulum and the Postoperative Ulcers in the Jepunum. Surg. Gynec. and Obst., 53:621, 1931.
12. Johnston, Lloyd B., and Renner, George. Peptic Ulcer of Meckel's Diverticulum: Report of 2 cases and a Review of the Literature. Surg. Gynec. and Obst., 59:198, August, 1934
13. Cobb, Donnell B. Meckel's Diverticulum. Peptic Ulcer Ann. Surg. 103:747, May, 1936.
14. Waugh, John M. and Herrell, Wallace E. and Crumpacker, Leo K. Peptic Ulcer in Meckel's Diverticulum Causing Intrinsic Intestinal Obstruction: Repor of 2 cases Surg. 2:385-391 (March) 1942.
15. Mason, J. M., and Graham, G. S. Ulceration of Aberrant Gastric Mucosa in Meckle's Dverticulum, a Source of Intestinal Hemorrhage. Ann. Surg. 96:893, 1932.
16. Rousseau, J. P. and Martin, A. G. M., III. Radiology 40:605 (June) 1943.

CYSTIC DISEASES OF THE LUNG

By DR. ERNEST C. REED

Prescott, Arizona

IT was not proposed to cover the entire field of cystic disease but to emphasize one type of which there had been three cases recently seen in the Veterans' Hospital. This type in the past has been variously called cystic degeneration of the lung; congenital cystic disease of the lung; pulmonary pneumocysts; bullous emphysema; cystic bronchiectasis, and more recently the names pneumocele and pneumatocele have been suggested.

The congenital etiology is open to much question because most patients give a history of pertussis; pneumonia; frequent colds or exposure to occupational dusts. The condition is seen in both sexes and the ages of reported cases varies from nineteen to seventy.

Pathologically there are found multiple, large thin walled sacs of various sizes, containing air and with tiny tortuous bronchial connections. Should a ball-valve mechanism develop in this connecting tube the affected cyst may enlarge rapidly and cause an acute episode of dyspnea as serious as sometimes occurs in spontaneous pneumothorax.

The symptoms commonly suggest asthma or asthmatic bronchitis; at least many of the patients have considered for a long time that they were affected with asthma. Diagnosis is made on the basis of x-ray film appearance, although the atypical asthmatic history; tendency to hyper-resonance and distant to absent breath sounds found on examination may suggest the condition. The roentgenogram shows an area of rarefaction usually at the apex or base of one or both lungs without any distinct border, and gradually blending with the areas where typical lung markings are seen.

The condition as shown by X-ray may remain stationary for many years or show varying rates of progression. Rupture of a cyst may result in collapse of a lobe or a lung and a film illustrating such a complication was shown. Empyema may follow such an accident. Diag-

Read before Yavapai County Medical Society, February 17, 1944.

nostic pneumothorax has been done to more clearly demonstrate the condition; but this procedure has its hazards and at least one death has been reported following such a procedure. Pneumothorax does not benefit the patient but only further embarrasses his respiration.

X-ray films were shown of three cases seen the past winter; one showed bilateral apical involvement extending down to about the second rib and apparently stationary over a period of two years; a second showed gradual progression of a less extensive condition over a period of three years and this patient was brought into the meeting for questioning. The third case was one of upper lobe involvement in a patient with advanced pneumonoconiosis; no progression over two years; the man had died of a brain abscess and autopsy had demonstrated the condition previously shown by X-ray. This lung, with one large thin walled air sac of tennis ball size and numerous smaller ones below it, was demonstrated by Dr. Wilkiemeyer, and photographs were shown of the lung as it appeared just after removal.

Pulmonary hypertensive heart disease may develop in the patient with advanced long standing cystic degeneration.

X-ray film showing induced pneumothorax was shown to contrast with the appearance of cystic degeneration and bring out the differential points. There is no known cure of the condition and patients so afflicted should try to avoid exposure to respiratory infections; seek a mild climate at not too high an tltitude and avoid more than moderate physical exertion.

PHYSICIAN-ARTISTS' PRIZE CONTEST

The American Physicians Art Association, with the cooperation of Mead Johnson and Company, is offering an important series of War Bonds as prizes to physicians in the armed services and also physicians in civilian practice for their best artistic works depicting the medical profession's "skill and courage and devotion beyond the call of duty."

Announcement of further details will be made soon by the Association's Secretary, Dr. F. H. Redewill, Flood Building, San Francisco, Calif.

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Editorials

The Bedside Manner

One of the most important characteristics of great men in medicine who have inspired and deserved our admiration and affection is the "bedside manner." The term as used here connotes depth of learning and sincere concern for the patient which is the only sound basis for confidence in a doctor. This "manner" has been and is seen at its best in consultation in the sick room for here the consultant observes meticulous consideration for his fellow practitioner, whose gues he is for the moment. There is nothing finer than this relationship in medicine.

Recently a Washington physician was quoted as saying that something should be done to prevent physicians from suggesting to patients that they have been carelessly or badly treated at the hands of a previous attendant. The implications in this statement are that doctors as a rule are unfair to each other. It can be safely said that rarely does a physician with malice of forethought disparage another practitioner in the presence of a patient. On the other hand, physicians have not always been as diligent as they might be to explain to a patient coming to them from another doctor that a sense of dissatisfaction or distrust may be based on misunderstanding or a faulty evaluation of results. Withholding a judiciously favorable comment on a colleague may really leave a physician open to serious criticism. For, as Robert Louis Stevenson observed, "One may

sit in a room and listen in silence while a friend is attacked and leave the room having more completely condemned his friend than if he had spoken."

It is not meant to suggest that malpractice should be condoned or defended but merely that physicians be extremely cautious and fair toward a previous attendant when they find themselves being credited with exceptional ability and judgment. Indiscretion is usually a boomerang might just as well be swung gently and carry a message of good will, to restore confidence to a doubting and unhappy patient, returning to give the sender a feeling of satisfaction in a deed well done.

Possibly the noise and confusion of the times may tend to dim the appeal of the Golden Rule. More, perhaps, than ever before we are called upon to be our brother's keeper. To serve our brother best, we must observe proper rules of conduct actively rather than passively. The Rule does not say we must not do unto others what we do not want them to do unto us but rather do unto others as we would have them do unto us. This, it would seem, is the keynote of the "manner" at the bedside as regards our fellow physicians. A fine "Bedside Manner" in the office, on the street, and even "off the record" is the medical man's attribute.

J. W. L.

Reprinted from *Medical Annals* of the District of Columbia, May, 1944.

Silicosis and the Arizona Occupational Disease Law

Elsewhere in this issue there will be found a supplementary report from the Subcommittee on Silicosis of the Arizona Medical Association, outlining the X-ray findings in this condition as they should be reported in order to conform to the definition of "silicosis" as contained in the Occupational Disease Law of Arizona.

Silicosis will become increasingly important to the medical profession of Arizona. Up to now, the number of claims have been few, because of the time limits set up in the law, by the anxiety of workmen to continue at work, if they can possibly do so, and by the need of employers for workers which has made them overlook physical conditions in many instances. With the ending of war, the curtailing of production in the mines, and the return of former workmen who have been serving in the armed forces, many present employees will be released. There will be a very natural desire on the part of such released workmen, if they have silicosis, to seek compensation for disability.

The medical profession should become well informed as to what constitutes disabling silicosis, and what is not recognized as such under our law. The report (see elsewhere in this journal) on the X-ray findings will be followed in subsequent issues by the other papers on silicosis, prepared by the several members of the Subcommittee. They will be a valuable reference work for all doctors in the state, because few will escape having to deal with this subject from some angle.

It All Began With the Pigeons

By now most people know the story of the pigeons and how they demonstrated the usefulness of microfilm. It happened in Paris when it was besieged in 1870 during the Franco-Prussian War. The French army headquarters at Tours wanted to get messages through the German lines. And so the photographer Dagron offered to take microphotographic equipment out of Paris in balloons. He escaped to Tours and sent messages back on film. Then he finally reached the army disguised as a wine merchant with his cameras in hollow wine barrels and he sent back to Paris over one hundred thousand messages on microfilm strapped to the quills of homing pigeons. (1)

That is how it began—the idea of transporting the written word in miniature and on film. But the possibilities of microfilming have not come to an end yet. About fifteen years ago a prominent New York banker conceived the idea of photographing bank checks as a means of keeping a permanent record. Within a short time the banking world, engineering concerns, insurance companies and numerous other types of organizations began to see the possibilities. And, now, medical record rooms and libraries have discovered microfilm, also.

The advantages to a library—and in this instance to a medical library—are many. For, with the use of microfilm the wealth of the resources in medical literature are at the disposal of any one possessing a film projector. Such

have been manufactured in various shapes and sizes. The one now in the Lois Grunow Memorial Library is a desk projector. The reader sits in front of the unit and turns the film at his convenience to read or study the information filmed. Illustrations such as photographs, charts, cut sections of tissue, and X-rays are clearly visible.

The purpose of microfilm is not to replace printed material, but to make available the information which would otherwise be difficult to obtain either because the original reference is oo valuable to lend, or because the information requested is too new to be reproduced in quantities available to all.

The Army Medical Library of the Surgeon General's Office in Washington, D. C., has initiated the Photoduplication Service. For a few pennies for postage this library will send a film copy of any article or book in that library. Incidentally, it is the largest and most complete medical library in this country and ranks with the most famous medical libraries of the world.

The resources of the Lois Grunow Memorial Library are open to any physician in Arizona. And so, with the microfilm service of the Army Medical Library and with the use of the microfilm reader in the Lois Grunow Memorial Library, the physicians of Arizona have at their disposal the wealth of the medical literature of the world.

LOIS GRUNOW MEMORIAL LIBRARY

Mary Elsie Caruso, Librarian.

(1) Litchfield, D. H. and Bennett, M. A. Microfilm reading machines. Special Libraries 34:15-20, January, 1943.

County Medical Society Life Memberships and Endowment Funds

In the July issue of the MISSISSIPPI VALLEY MEDICAL JOURNAL (Quincey, Ill.) Swanberg advocates the establishment of Endowment Funds and Life Membership plans in all but the smaller County Medical Societies. "This plan affords the member an opportunity of paying his full dues during his most productive years and while his income is greatest, thus avoiding the burden of dues later in life." Since Life Membership fees can be declared a professional expense when filing income taxes their actual cost is not great. The present era of high incomes and high income taxes thus provides an ideal time for making an investment in one's County Medical Society. The actual cost of a Life Membership is considerably less than the amount paid since 27 to 57% (depending on the surtax net income) represents tax savings. If Life Membership fees are invested in war bonds and placed in an Endowment Fund, it will further help the government finance the war, a patriotic undertaking of which every loyal American physicial wants to be a part.

Swanberg also states that "physicians, as a class, have not been generous with their contributions to medical organizations, and it is high time efforts were made to correct this situation." He feels that "the physician should give more consideration to making direct contributions or remembering his County Medical Society in his will; such contributions, at this time, represent substantial tax savings so that the amount contributed costs considerrably less than the actual contribution."

HIGHLIGHTS IN THE HISTORY OF NATURAL QUININE

(Digest of an article by Norman Taylor, in the July, 1943, Scientific Monthly. Mr. Taylor is Director of the Cinchona Products Institute, New York).

Natural quinine is found exclusively in the bark of cinchona trees which grew originally in South American countries. Its first use in the treatment of malaria is not consistently recorded in history. How quinine arrived on the European continent is also a subject of conflicting stories. However, it is first mentioned in European medical literature by a Belgian, Herman van der Heyden in 1643.

For the next 200 years quinine was extracted from cinchona bark on a large scale to be used as the only effective anti-malarial drug. England and Holland in the 1850's attempted to start cinchona plantations respectively in India and Java, because the cinchona supply was dwindling in South America. Their efforts were unsuccessful because the type of cinchona seed they used produced a tree with a very low quinine content, too low to produce practically.

In the romance-packed history of quinine, the story of Charles Ledger is outstanding. After a series of adventures, Ledger selected

a cinchona type tree which he considered to have the highest quinine content. One pound of seed from this type, which now bears the Ledger name, was sold to the Dutch Government cinchona plantation in Java in 1865. With this pound of seed the Dutch established plantations which in 1941 produced over 1000 tons of quinine. Java had become the source of nearly all the world supply of quinine.

Since the seizure of Java by Japan in March, 1942, strenuous efforts have been made by this country to conserve the quinine supply on hand and to establish new cinchona growing areas.

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COUNCIL MEETING

The Council convened at the Westward Ho Hotel, Phoenix, on July 16, to transact their summer's business prior to fall activities. Present were: Drs. Dan L. Mahoney, Walter Brazie, Chas. P. Austin, Frank J. Milloy, C. E. Yount, O. E. Utzinger, E. Payne Palmer, F. W. Butler, J. Newton Stratton, Robert S. Flinn, Jesse D. Hamer. Absent were: Drs. Hal W. Rice and Geo. O. Bassett.

Business transacted: 1. Dr. Geo. O. Bassett was elected Chairman of the Council to serve for 1944-1945.

2. The Agricultural Workers Health and Medical Association Hospital (Burton-Cairns) at Eleven Mile Point in Pinal County was reported closed by order of the Board of Directors, San Francisco office, as of June 30. The Report is printed in this issue of Arizona Medicine.

3. Dr. Milloy reported on the Associated Hospital Service of Arizona, stating that the organization was still existent but had been inactive for lack of some one to push it. He further stated that a young man was now inter-

ested in promoting the plan and that something might come of it. The insurance plan adopted by the Associated Hospital Service is in line with the Blue Cross and can become a member by meeting a specified membership, and showing a $5,000 reserve which latter the association in question now has. No formal action was taken by the Council, the Committee on Medical Economics already studying such plans.

4. President Mahoney reported to the Council on various matters. The new program of Vocational Rehabilitation, as discussed at the Annual Meeting, was again before the Council for final clearance A list of physicians in the state certified by various specialty boards was approved by the Council for submitting to the Direcor of Vocational Rehabilitation for appointment to an Advisory Committee. Our members are urged to accept appointment to this Advisory Committee if selected in their regular fields of specialties by the State Agency for this purpose. The matter of appointment of the medical member to the State Board of Institutions for Juveniles was also discussed. The term of Dr. M. W. Westervelt as a member of this board expired last February 1. The law requires of the Governor that he request the Council of the Arizona State Medical Association a list of three names from which he shall select one to present the Senate for ratification. Such a list was submitted the Governor at his request last spring when the special session of the legislature was sitting but appointment is still pending.*

5. The Report of Harold W. Kohl, M. D. (Lt. Colonel, M. C.), was read and accepted. The report appears elsewhere in these columns. Dr. Hamer, as alternate-delegate and who was also in attendance, reported verbally on interesting discussions and actions of the House of the A.M.A. See the A.M.A. Journal for June 24 for complete Minutes of the House.

6. The Council confirmed numerous appointments to the various committees as submitted for its consideration by President Mahoney. A Committee roster appears in other columns of this issue.

7. The Council voted to continue the present series of Health Education radio programs over the Arizona Network, and considered expanding its programs beginning with January next.

8. It was voted by the Council, Dr. Chas. P. Austin, Chairman of the Committee on Scientific Assembly, opening the matter for discussion, that a group of instructors from some oustanding and nearby medical college be invited to present the scientific program for the Annual Meeting as was done with marked success last year. Dates for the Annual Meeting, subject to the approval of the host (Pima) society, for the meeting for 1945 were tentatively set for April 28 and 29. The meeting place is to be Tucson as the sessions alternate between Phoenix and Tucson under By-law provision.*

9. A report from Dr. Chas. S. Smith, Chairman of the Committee on Procurement and Assignment was heard and accepted. The report appears elsewhere in this issue.

10. Dr. Jesse D. Hamer, chairman of the Committee of Public Policy and Legislation, reported on pending national and possible state legislation relating to the practice of medicine. Of this, the membership will hear more at a later date.

11. An informal report was heard from the treasurer, Dr. C. E Yount, showing a paid-up membership of 284 at this time.

Adjournment was 3:30 P. M.

Frank J. Milloy, M. D.,
Secretary

REPORT OF THE DELEGATE TO THE AMERICAN MEDICAL ASSOCIATION

To the members of the Arizona State Medical Association:

The 1944 session of the House of Delegates of the American Medical Association at the Palmer House, Chicago, was attended by your delegate and by Dr. Jesse Hamer, alternate delegate and delegate-elect. It is well that it was so because your delegate found himself obligated to leave Chicago for his station in Florida on Wednesday, June 14th, before the

* Dr. F. W. Butler, of Safford, was appointed on July 30 by the Governor to serve on this board.

* A group of Instructors from Baylor University (Texas) was present at the Scientific Program.

adjournment of the House. A supplementary report will be submitted to the Arizona Association by Dr. Hamer, covering business transacted and not referred to in this report.

Dr. George Dock was awarded the Distinguished Service Medal by the House on the second ballot. Other names presented this year to be voted upon, together with that of Dr. Dock, were Dr. Isaac A. Abt and Dr. Simon Flexner. Dr. Dock is noted for his work on the pathology of malaria and dysentery, protozoan diseases of the blood, pernicious anemia, the ductless glands, and hookworm. He is 84 years of age. Dr. Dock has been on the teaching staff at the University of Pennsylvania School of Medicine, Texas Medical College, University of Michigan Medical School, Washington University, and University of Southern California School of Medicine. He was vice president of the International Medical Congress at Moscow in 1897 and at London in 1913. He at present resides in Pasadena.

Dr. James Paullin, the retiring president of the American Medical Association, in his address stated among other things that some state societies are at odds with the House of Delegates on broad policies. Many comments are heard among the Doctors of America that the American Medical Association does not truly represent the views and desires of American doctors. Dr. Paullin conjectures that the open disagreement of state societies with the American Medical Association is largely responsible for that undercurrent of opinion. Generally speaking, your delegate cannot help but feel, after five years in the House, that state societies must continue to stimulate the national body to deep thought in the consideration of the changing way of things in this country. The American doctor must show the way in the post war reconstruction period where Medical matters are concerned. The County and State Societies are those integrals which must be the meeting places for American physicians to contemplate the current problems of organized medicine, to conceive ideas based on personal contact experience with existing trends, and to formulate plans and suggestions to be transmitted to the American Medical Association. The parent body must then in turn be sympathetic and understanding with her component societies, giving careful consideration to ideas and suggestions emanating therefrom, in order that our future may be secure as free physicians, and as a healthy national organization.

The California Delegation introduced a resolution approving and endorsing the creation of the United Public Health League was reported on unfavorably by Committee and was not adopted by the House of Delegates.

The California Delegation also introduced a resolution that certain of the officers of the American Medical Association, including the editor, Dr. Morris Fishbein, be replaced and that Dr. Olin West be commended "for his long valuable and faithful service to American medicine, expressing the gratitude of the profession for that service, assuring him of our affection and promoting him to the office of Secreary Emeritus for life." That resolution was reported on unfavorably by Committee and the House voted not to adopt it, by a vote of 144 to 9.

The Reference Committee on Medical Education reported upon the present status of enrollment in our medical schools throughout the nation. Since there is no deferment by the Selective Service of students not yet in medical schools, and since the Army Specialized Training Program has been curtailed, it appears that approximately 43% of the necessary enrollment in medical schools, to continue a normal production level, must be obtained from the physically disabled and from women. The Council on Medical Education and Hospitals is in constant touch with problems and developments along this line under the able direction of Dr. Ray Lyman Wilbur of Stanford University.

A Council on Post War Planning was established by vote of the House of Delegates to weigh the problems of the post war period including those of some 55,000 physicians who are now in the armed services and who hope to be returned to their several communities with the advent of peace. Many will desire residences, refresher courses in their respective specialties, and aid in obtaining suitable locations in which to resume practice. This Council is charged with the huge and important task

of presenting adequate solutions to these problems.

American Medicine now as ever is meeting all challenges and acquitting itself in a highly commendable manner.

Your delegate is grateful for having had the opportunity to serve you during the past five years and has considered that service an honor and a privilege.

H. W. KOHL

REPORT ON PROCUREMENT AND ASSIGNMENT OF PHYSICIANS

To the Council,
Arizona State Medical Association:

A report of the conference on P&AS held in Chicago June 13, 1944 is herewith presented for your consideration.

Insofar as Arizona is concerned, there are hardly any physicians, who have not previously been rejected for physical reasons, whom we can declare available. At the present moment I do not know of any Arizona physician who is being considered for availability. According to the last official figures given me Arizona has approximately 195 physicians in the Armed Forces. I have not had time to check this list thoroughly, but the above figure is approximately correct.

There are at present 54,096 physicians in the Armed Forces. As a result of the Army cut in its specialized training program, there will be in 1945 enough Army students to fill only 28% of the places; the Navy will supply 31%, and 41% presumably will be available for women and physically disqualified men.

As a result of Selective Service policy of eliminating deferments of pre-medical students after July 1, it is assumed there will result a shortage of physicians available for the civilian population. Of approximatetly 6400 places to be filled in 1945, 3400 can be filled by the Armed Forces, leaving about 3000 to be filled from the ranks of women and physically disqualified men. Half of the latter number, according to present experience, will obviously be left unfilled unless there is some modification of existing policies.

Dr. Lahey summarized the Board's position as follows: (1) Selective Service should defer pre-medical students and (2) P&AS is unwilling to support, at the present time, a program calling for the return of soldiers on active duty to fill medical school vacancies.

Dr. Barton, Executive Officer, P&AS, explained that with more than 54,000 physicians in the Army and Navy, there are few left who can still be considered available. Since one of P&AS dual responsibilities is civilian care, close scrutiny must now be given to each individual case before an availability is granted. Dr. Barton explained that the Central Office is reviewing all availabilities in order to make sure that a danger point is not reached. He emphasized also that equitable distribution of practitioners is now a major responsibility of the Service and that although relocations are difficult to effect in many cases, "We must do what we can with whatever weapons we have."

There are at present approximately 400 physicians in practice in Arizona; insofar as I know, with the possible exception of Grand Canyon, Arizona, there are no areas in the state suffering from lack of medical care.

With the changing of the status of many physicians; some coming into the state, others leaving the state, I am finding it very, very difficult to keep my "fingers" on each physician. My work could be made much easier if the Secretary of each county medical society would report to me any change in the status of any physician residing in his county. Washington is demanding more than ever before a report on EACH physician in Arizona, and for me to be able to send an intelligent report —one that is authentic, I seek the aid of every physician in the state to this end, viz: report any "newcomers" or any leaving the state direct to me.

I am truly most grateful for the cooperation extended me by the members of our statewide committee and the Council of the Arizona State Medical Association.

Respectfully submitted,

CHARLES S. SMITH, M. D.
Chairman State of Arizona, Procurement and Assignment Service for Physicians.

REPORT OF BURTON-CAIRNS HOSPITAL, AGRICULTURAL WORKERS HEALTH AND MEDICAL ASSOCIATION. ELEVEN MILE POINT, ARIZONA

As a report to the Council on the Burton-Cairns Hospital at Eleven Mile Point, I quote from the minutes of the meeting of the directors' meeting of the Agricultural Workers Health and Medical Association held at Berkeley, California on June 9, 1944.

"The disposition of the Burton-Cairns Hospital was then discussed, and Dr. Mott suggested that since there have been very few patients in the hospital with the cost averaging about $20 per patient day during May, the hospital should be closed. It is hoped that arrangements could soon be made for another agency or group to run the hospital until such time as it would again be needed by the Agricultural Workers Health and Medical Association.

Dr. Thomas agreed that the cost at present was uneconomically high with only fourteen patients at present in the hospital.

Mrs. Montague stated that arrangements could be made for the patients now in the hospital who needed extended hospitalization. It was the general opinion that it would be best to lease the hospital to a non-profit organization rather than to private practitioners. Dr. Mott stated that there is a bill before Congress appropriating ten million dollars for tuberculosis control and that, if it should pass, it is highly probable that some group could make use of some of these funds in order to operate this hospital as a tuberculosis center."

It was the duty, as I understood it for Mrs. Helen Montague, General Manager of the Agricultural Workers Health and Medical Association for Arizona, to supervise the closing of the hospital. Her statement as to such closing follows:

"According to the instructions by the Board of Directors of the Agricultural Workers Health and Medical Association, the hospital (Burton-Cairns) was closed June 30, 1944. The patients that needed further hospitalization were transferred to hospitals in Phoenix. A full time caretaker has been retained."

The above covers the most recent action in relation to the Burton-Cairns Hospital under the management of the Agricultural Workers Health and Medical Association in Arizona.

Signed

CHARLES A. THOMAS, M. D.
Member Board of Directors for Arizona.

July 12, 1944.

Staff Meetings

AMERICAN COLLEGE OF CHEST PHYSICIANS

At the annual meeting of the American College of Chest Physicians held at Chicago, Ill., June 10-12, 1944, Dr. Charles S. Kibler, Tucson, was re-elected as Regent of the College for District No. 12 comprising the states of Arizona and New Mexico, for a term of three years.

Dr. Howell S. Randolph, Phoenix, was elected Governor of the College for a term of three years.

Physicians from Arizona who attended the meeting were:

Dr. Hilton J. McKeown, Phoenix
Dr. Howell S. Randolph, Phoenix
Dr. Benson Bloom, Tucson
Dr. Charles S. Kibler, Tucson
Capt. Peter Amazon, M.C., Coolidge.

MARICOPA COUNTY MEDICAL SOCIETY

Monday Evening, May 1, 1944, 8 P. M.

Scientific Program

1. The Pathogenesis of Silicosis:
   Maurice Rosenthal, M.D.
2. The Classification of Silicosis—from a Radiological Viewpoint:
   W. Warner Watkins, M.D.
3. Some Clinical Aspects of Silicosis:
   Louis B. Baldwin, M. D.

GOOD SAMARITAN HOSPITAL

(Phoenix)

APRIL 24, 1944

Program

I. An Unusually Large Ovarian Cyst
   Dr. Louis P. Lutfy
II. Case of Dissecting Aneurysm of Aorta With Rupture
   Dr. John W. Pennington

MAY 22, 1944

I. A Questionable Case of Periarteritis Nodosa
   Dr. Norman A. Ross
11. A Case of Chirrosis of the Liver
   Dr. Robert S. Flinn

JUNE 26, 1944

1. Angioma of Ovary in 6-Year-Old Girl
   Dr. Robert T. Phillips
II. Basal Cell Carcinoma in 16-Year-Old Girl
   Dr. Louis G. Jekel
111. Case of Coronary Occlusion
   Dr. Ben Pat Frissell

ST. JOSEPH'S HOSPIAL STAFF

(Phoenix)

MAY 8, 1944

I. Review of Literature on Treatment of Burns
   Resident Staff

II. Idiopathic Purpura Haemorrhogica Fulminans
Dr. A. E. Cruthirds

JUNE 8, 1944

I. Present Status of Pencillin Therapy
Dr. R. Jannett

II. Carcinoma of Lung With Metastates in Brain
Dr. Robert H. Stevens

Clinical Pathological Conferences

F. W. 58-year-old white woman.

March 15, 1944: The patient was perfectly well until three weeks ago when she began to develop an aching pain between the shoulders which she treated with a mustard plaster. Five days later jaundice developed which has percisted to the same degree since. Associated with this there has been nausea and vomiting, at first ten to eleven times a day and then two to three times a day, which has continued. The stools have been light colored on a few occasions, though they are usually brown. The dull ache between the shoulders has recurred several times a day since its onset, and for the past four days there has been an almost constant dull ache to the right of the umbilicus, occasionally radiating down to the left lower quadrant. Eating greasy foods has always made her "billious."

In December, 1943, she passed part of a "tape worm"; had lost 30 pounds of weight previous to this, and began to develop boils. She believes she has lost a little weight again with the present illness. With the tape worm episode she was given some medicine which she took for a few weeks, the nature of which is not known.

Physical Examination: Temperature 98.6; pulse 110; respirations 40; blood pressure 114/64. The patient is a markedly jaundiced white female lying in bed in moderately acute distress, complaining of pain between the shoulder blades and in abdomen. Sclera are jaundiced. Edentulous. Heart normal. Lungs: moist rales at both bases with bronchial breath sounds at right base posteriorly. Abdomen: distended and rounded, ++ rigidity in right upper qaurant, +++ tenderness in right upper quadrant, and ++ in left upper quadrant and + in right lower quadrant; ++ rebound tenderness right upper quadraut, left upper quadrant, and right lower quadrant. Peristalsis is absent. Liver percussible 2 cm. below right costal margin. No masses. Percussion note is hyperresonant with no fluid wave. No costovertebral angle tenderness. Rectal and pelvic negative except for slight tenderness high in cul-de-sac.

Blood: Hh. 83%, Wbc. 36,850; P. 92%, L. 8%.

Urine: 250 cc., dull greenish in color. Albumin O, sugar O, bile +.
Sp. Gr. 1.020.

Pottenger suction started and patient feeling better with less abdominal pain and tenderness.

3-16-44. 3:00 a.m. Temperature 100°. Patient feeling a little better.
7:45 a.m. Temperature 102°. Pulse 100; less pain but rigidity persists.
1:30 p.m. Patient cold and clammy; semistreporous. Cardia rate 100. Blood pressure 0/0. No response to caffeine sodium benzoate. Patient died in a few minutes after this.

NPN 53. Urine diastase normal. Icteric index 51.

A/G 3.6/2.1. Blood diastase less than 60. Prothrombin content less than 5%.

Blood Wassermann and Kahn—negative.

DR. WILLIAM DOCK: This 58-year-old woman entered the hospital on March 15th, three weeks after the onset of an aching pain in the back, between the shoulders. While such a pain most often is due to arthritis, it might have been due to an upper abdominal or to a mediastinal lesion. A few days later jaundice was noted, but in the two weeks that followed did not deepen, remaining constant. This is most unlikely to have been obstructive jaundice, since it did not rapidly deepen, and even the jaundice of acute hepatitis, which is suggested by the intense nausea, should have darkened during the first two weeks. The pain of acute hepatitis or yellow atrophy may be typically biliary, with gall-bladder tenderness and is seen not rarely in fatal cases, although it is rare in the benign or common forms. The shift of the pain into the para-umbilical region and left lower qaudrant suggests carcinoma or acute inflammation of the pancreas.

The patient had no significant past illness except for recent furuneulosis and treatment for tape-worm. The latter seems too long ago to explain the liver disease, the boils may have been a source for visceral abscesses with hemolytic jaundice, but in the absence of chills and fever this is most improbable.

The patient on entry was afebrile, but the pulse and respirations were so rapid as to suggest shock. The jaundice is definite, the liver slightly enlarged and there are the classical physical signs of peritonitis. This certainly suggests suppurative biliary tract disease, with a terminal peritonitis, possibly but not necessarily due to perforation or rupture of the duct or gall-bladder.

The high white count also fits into the picture of sepsis, but in leptospiral jaundice and

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Presented before Arizona State Medical Association, Phoenix, April 15, 1944.

rarely in acute yellow atrophy there is marked leucocytosis. The description of the stools, and the relatively low icterus index of 51 seem to rule out obstructive biliary tract diease. There may, however, be pylephlebitis, arising in a buried appendix perhaps, or an ascending biliary tract infection without obstruction. The normal diastase makes pancreatitis a remote possibility, but does not rule out a cancer of the body of the pancreas. The latter often is a cause for pain and nausea. The severity of the nausea at the onset of jaundice, and the very low prothrombin fit better with an acute hepatitis.

The fever became apparent, shock more profound and death occurred, presumably from peritonitis, in 24 hours. Peritonitis is not a rare cause of death in cirrhotics, but this patient has normal serum protein and no leucopenia, which rule out cirrhosis as far as I am concerned in a case with the other findings here present.

My first bet would be acute hepatitis, possibly due to leptospirosis, and terminal peritonitis. But this diagnosis is not confidently held, and asceneding biliary tract disease or pylephlebitis are good probabilities. There is no real evidence of cancer.

Anatomical Diagnosis

DR. EDWARD M. BUTT: Anatomical Diagnosis: Carcinoma of Head of Pancreas.

STAFF MEETING, ST. JOSEPH'S HOSPITAL

The case is that of an 8-year-old child, who first entered the hospital because of secondary optic atrophy of the left eye. Her history is that she lived with a sister who had died of pulmonary tuberculosis. Birth was normal and she had a normal infancy. Pertussis at 2 years followed by frequent colds ever since. Chicken pox at 3; last summer had a headache over the left eye, vomiting, fever and extreme nervousness for several days and ever since, she has had this type of headache.

For about two months, she could not see out of the left eye at all.

X-rays of the head show sinuses are well developed and are transparent; the selle is irregular in shape and possibly slightly enlarged. Optic foramina are normal in size and shape and are alike on each side. Chest—mitral configuration of the heart, increased hilar densities; apparently not a tuberculous reaction.

Two years later she again entered the hospital unconscious, vomiting and choking. Mother states that that morning, the patient complained of burning sensation over her eyes and was coughing up mucus. After about an hour, the mother found her in bed, unconscious and called the doctor. Previous to this, the patient had been in apparently good healh. As to drugs, the patient has had only aspirin. Loss of sight in the left eye.

Comparison of X-ray with film previously taken shows the same configuration of the heart; not much change in the hilar densities. On the skull films, there is no definite change in the bone densities and no change in the sella. Laboratory findings—blood sugar 180 mg.

Progress notes state that the child entered the hospital in a comatose condition with flaccid extremities. Shortly after entering, the temperature shot up—from 99 to 104° and cyanosis and convulsions developed. Blood pressure varied from 130/100 to 110/80. Cyanosis and heart failure that afternoon resulted in death.

DR. R. LEE FOSTER: The radiologist becomes accustomed to aproaching the diagnosis of a case with the X-ray findings as the first clue. Right or wrong this habit develops since the X-rays are practically always his first knowledge of a case, and all too frequently the only information he is given with which to work.

In this case the positive X-ray finding which no one has disputed is a mitral configuration of the heart with increased hilar densities and the latter the radiologist did not believe due to a tuberculous reaction. The outstanding condition which would produce a mitral configuration of the heart in a child of this age would be a rheumatic fever. However, when we consider the other X-ray finding, namely, an irregular and possibly enlarged sella, it is hard to fit it with a rheumatic fever. Although rheumatic fever can produce focal vascular lesions in the brain they are rare and would hardly cause sellar changes. There is, however, a disease which may simulate rheumatic fever, often complicates it and is even considered by some as an atypical form of the same disease. I'm speaking of Sydenham's Chorea.

Now if we consider this possibility in the light of the clinical history, physical findings, and course of this patient's illness, we find that it fits rather nicely in most respects with the acute form of Sydenham's Chorea with brain lesions and optic neuritis.

The disease is most frequent between ages 5 to 15 years and occurs 2½ times as frequently in girls as boys. Rheumatic fever and other acute infections predispose to it and may accompany it. This patient is a female, 8 years of age, and had pertussis at the age of two years followed by frequent colds since.

The onset may be sudden or very gradual. There is usually headache, anorexia, constipation and occasionally vomiting. Fever is usually low grade. The severity of the disease varies greatly. The cardinal symptoms are spontaneous, usually ataxic, movements, weakness and usually phychic changes. If we consider the "nervousness" spoken of in the first attack

as being possibly mild choreiform movements, the patient has all of these things.

Although a fatal termination is rare, it does occur in the acute cases as about 1 to 2% and is most often due to cardiac complications.

Brain complications are not infrequent and in fact the pathological lesion is a low grade meningo-encephalitis, and cerebral congestion with hyperemia and numerous thromboses are usually found through the brain and meninges. Embolism of the central retinal artery may occur and an optic neuritis is not at all infrequent. Gowers states that the swelling from the optic neuritis may occasionally become large enough to simulate a cerebral tumor. Hyperpyrexia, vomiting and any other symptoms of intracranial disturbances may occur.

Here then, if we are not too insistent on the presence of violent choreiform movements as a necessity to diagnosis, is a case which could well be an acute attack of chorea with an optic neuritis which recovered and recurred two years later as another acute attack, again with optic neuritis, and progressing rapidly to a fatal termination due to cardiac failure from cardiac complications with intracranial involvement as a contributing factor.

I present that as a possible diagnosis.

ATROPHY OF THE OPTIC NERVE

DR. CHARLES N. PLOUSSARD: This affection occurs either (1) as a *primary* disease (simple, gray, non-inflammatory or progressive atrophy) or (2) *secondary* to some other affection of the nerve or retina (neuritic, postpapillitic, or inflammatory atrophy); in the latter class belon galso *retinitic* and *choroiditic* atrophy.

Symptoms

There are reduction in the acuteness of vision, concentric contraction or irregular or sector-shaped peripheral defects of the field first for colors and then for form, diminution in the light sense, sometimes *scotomata*, and *color blindness* (first for green, then for red, then for blue). These symptoms tend to progress and end in complete *blindness*.

OPHTHALMOSCOPIC SIGNS depend somewhat upon whether the type is primary or secondary:

Primary Atrophy: The *disc is white, grayish*, or bluish-white, its *edges are sharply defined* and regular, its size is somewhat diminished, and it presents a saucer-shaped excavation; the *lamina cribrosa* is often seen very *plainly;* the minute vessels of the disc have disappeared; the retinal vessels may appear normal or the arteries may be diminished in calibre.

Postpapillitic atrophy: The *disc is dense white* or grayish in color, sometimes with a bluish tint, its margins *irregular* and somewhat *hazy*, its minute vessels lost and it is covered by connective tissue resulting from the organization of the previous exudate; on this account the lamina cribrosa is hidden; the retinal arteries are narrow, the veins normal in size or distended and generally tortous and both sets are apt to be enclosed by *white lines.*

Retinitic and Choroiditic Atrophy: The *disc* has a grayish-red or *yellow*, waxy appearance, its outlines are somewhat *indistinct*, the *vessels* are exceedingly *narrow* and many disappear entirely, and the retina presents evidences of the antecedent choroiditis or retinitis.

After a time, the differences in the appearances of simple and postneuritis atrophy become much less marked.

It should be borne in mind that the *disc varies in color in health* and may appear atrophied as the result of congenital or senilepeculiarities, although vision is normal and the field perfect; hence the diagnosis in many cases cannot be made from the ophthalmoscopic signs alone, especially when these signs are not pronounced.

ETIOLOGY: Simple atrophy is frequently due to spinal diseases, especially locomotor ataxia, developing as an early symptom in one-third of the cases of this affection. It is common also in affections of the brain, especially disseminated sclerosis, general paralysis of the insane and tumors. It may also be due to syphilis, malaria, diabetes, acromegaly, impaired nutrition, arteriosclerosis, and certain poisons (including wood-alcohol). Occasionally it is hereditary, and in some cases no cause can be found. Hereditary cases occur in young adult males, involve the papillo-macular bundle, are accompanied by central scotoma, and the affection is known as *Leber's Disease.*

Secondary atrophy follows choked disc, descending neuritis, ecc pigmentary degeneration of the retina, and embolism and thrombosis of the central artery; it may also be consecutive to choroiditis retinitis, glaucoma, hypophysis disease, and orbital inflammations. It may result from injury to the optic nerve due to fracture of the orbital canal, following a blow or other violence; in such cases the atrophy does not show itself for a number of weeks, though reduction of vision and contraction of the field or even blindness ensues immediately.

Diagnosis

(1) Brain tumor
(2) Brain abscess
(3) Diabetes.

DR. VIRGIL TOLAND: It is unfortunate that the findings in the right eye are not given for this is a condition where the ophthalmologist could definitely help the clinician in establishing a diagnosis. From the oculist's viewpoint there are three likely possibilities depending upon the findings in the other eye. The first is a retinoblastoma. If the right eye was negative, the left eye might have contained this

tumor, which could have been confused with an optic atrophy, and metastasized along the optic nerve into the brain accounting for the cerebral symptoms. However, cases of retinoblastoma rarely live to be eight years old. The second possibility is a Foster Kennedy syndrome. Primary optic atrophy in one eye, and papilloedema in the other. This syndrome is pathognomonic of frontal lobe tumors, the tumor arising in one frontal lobe presses on the corrseponding optic nerve causing atrophy then keeps on growing until it causes increased intracrainial pressure and a choked disc in the other eye. The third condition is a Juvenile Form of Amaurotic Family Idiocy. If the opposite eye also showed a primary optic atrophy and the patient had developed symptoms of idiocy then this could have been the cause of death.

Pathological Report

DR. TERESA R. MORAN: *Brain:* The scalp is incised and the calvarium removed. There is an increased amount of sub-arachnoid fliud present. The confolutions are somewhat flattened and the sluci are shallow. On lifting the anterior lobes considerable hemorrhage is found present on the lower surface. This extends into the pituitary region and around the base of the brain. The sella turcica is broadened. The cliniod processes are absent and a large growth occupies the sella and extends around the pons and is seen to involve the left optic nerve. This growth is light brown in color and markedly edematous. It is in the region of the optic chiasma and the pituitary gland. No pituitary gland is found present. The brain is preserved for section after fixation.

Anatomical Findings

1. Ependyma of brain involving
   (a) frontal lobe
   (b) optic chiasma
   (c) optic nerve
2. Cerebral hemorrhage intraventricular and subarachnoid.
3. Passive hyperemia of viscera.

THE AMERICAN CONGRESS OF PHYSICAL THERAPY

Will hold its twenty-third annual scientific and clinical session September 6, 7, 8 and 9, 1944, inclusive, at the Hotel Statler, Cleveland, Ohio. Rehabiliation is in the spotlight today, Physical Therapy plays an important part in this work. The annual instruction course will be held from 8:00 to 10:30 a. m., and from 1:00 to 2:00 p. m. during the days of September 6, 7 and 8. The scientific and clinical sessions will be given on the remaining portions of these days and evenings. All of these sessions will be open to the members of the regular medical profession and their qualified aids. For information concerning the instruction course and program of the convention proper, address the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, 2, Illinois.

Woman's Auxiliary

STATE AUXILIARY OFFICERS AND COMMITTEE CHAIRMEN

PRESIDENT ..... Mrs. James H. Allen
Prescott
PRESIDENT-ELECT ..... Mrs. Paul H. Case
Phoenix
FIRST VICE-PRESIDENT ..... Mrs. W. Claude Davis
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Cancer Project: Mrs. L. D. Beck, Phoenix
Legislation: Mrs. C. E. Patterson, Tucson.
Public Relations: Mrs. George L. Dixon, Tucson.
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Historian: Mrs. George B. Irvine, Tempe.
War Service: Mrs. Jesse D. Hamer, Phoenix.

MRS. JESSE D. HAMER, NEW NATIONAL PRESIDENT-ELECT WOMAN'S AUXILIARY TO THE AMERICAN MEDICAL ASSOCIATION

The Arizona Auxiliary is proud and happy to report that Clarice Hamer, wife of Dr. Jesse D. Hamer, Phoenix, is the new national president-elect of the Woman's Auxiliary to the American Medical Association. She will take office in 1945.

In accordance with the traditional procedure of the auxiliary, it creates the office of president-elect as well as four vice presidents.

Election was held during the national convention in Chicago June 12 to 15.

Mrs. Hamer, a registered nurse, has been as active member of the Medical Auxiliary for a number of years and in 1940 became Arizona's state president and a member of the national board. The following year she was made national chairman of legislation, and last year served as a national director.

Other new national officers are: Mrs. David W. Thomas, Lockhaven, Pa., president, who succeeds Mrs. Eben J. Carey of Wauwatosa, Wis.; Mrs. Eustace A. Allen, Atlanta, Ga.; Mrs. J. Howard Hornberger, Roebling, N. J.; Mrs. Arnold Duemling, Ft. Wayne, Ind.; Mrs. David Berg, Helena, Mont.; the four vice presidents; Mrs. A. A. Herold, Shreveport, La., secretary; Mrs. Harold F. Wahlquist, Minneapolis, treasurer; and Mesdames Carey James Simonds (Chicago), W. W. King (Denver), Roscoe E. Mosiman (Seattle), Luther H. Kice (Garden City, L. I.), directors.

MRS. T. A. HARTGRAVES,
Publicity Chairman.

CONVENTION REPORT
WOMAN'S AUXILIARY TO THE AMERICAN MEDICAL ASSOCIATION

Chicago, Illinois

June 12-15, 1944

The twentysecond annual meeting of the Woman's Auxiliary to the American Medical Association was held at the Knickerbocker Hotel at Chicago, Illinois, from June 12-15, 1944. The president, Mrs. Eben J. Carey of Wauwatosa, Wisconsin, presided over all the sessions.

Monday, June 12th was given over to registration of delegates and guests and the pre-convention board meeting.

The first general session was called to order at 10:00 o'clock on Tuesday morning. The meeting was opened in the usual manner Mrs. M. A. Nix of Illinois gave the Address of Welcome. The Response was given by Mrs. Asher Yaguda of New Jersey. Officers and chairmen of standing committees then gave their routine reports. Mrs. Carey's report stated that the Advisory Council had asked the auxiliary to lend its assistance and support to three major projects during the past year, and that most of the organized auxiliaries had participatetd in all three so far as was possible. The three projects were: Promotion of the Nurse Cadet Corps; the national registration of all graduate nurses and to help defeat the Wagner-Murray-Dingell Bill. The national registration of graduate nurses was postponed at the last minute but the auxiliary members throughout the country were prepared to render whatever assistance necessary to the carrying out of this program in cooperation with the State Nurses' Associations. The chairman of legislation stressed the importance of being ever on the alert regarding medical legislation and mentioned briefly some of the bills now pending. The value of Hygeia was greatly emphasized by the chairman and by Dr. Herman L. Kretschmer as well, who suggested that every doctor shoud subscribe to two issues, one for his home and one for his office.

The War Service Committee, which was created at the annual meeting in 1943, gave an excellent report of the war work done by auxiliary members throughout the United States. They are taking an active part in almost every type of war work which is being done. In many instances they are furnishing the leadership for important projects.

An "In Memoriam" service was conducted. The recorded showed a loss of one hundred thirty-four members during the past year.

The main business transacted during the convention was the adoption of a new Constitution and By-Laws. There were many minor revisions, but the four major revisions concern the membership and duties of the Board of Directors; the standing committees; election and the setting up of a state presidents' conference.

Due to the length of time taken up in considering the Constitution and By-Laws, the state presidents' reports were not read, but will be printed in full in the post-convention Bulletin.

Delegates from almost every organized auxiliary in the United States were present. The total registration was five hundred twenty.

Inspiring talks were given by Miss Reese of Chicago, concerning the Recruitment of Student Nurses; by Vice Admiral Ross T. McIntyre, Surgeon General, U. S. Navy, on "Women and the War"; Dr. Kretschmer, President-elect of the American Medical Association; Dr. James E. Paullin, President of the American Medical Association; Dr. Morris Fishbein, Editor of the Journal of the A.M.A. and Hygeia and Dr. Rollo K. Packard of Chicago, on the subject of the need of some form of prepaid medical care.

The social activities consisted of a tea honoring Mrs. Eben J. Carey, the president, Mrs. David W. Thomas, president-elect and the National Board; a luncheon honoring Mrs. Carey; a luncheon honoring the past presidents and a tour of exhibits and dinner at the Museum of Science and Industry.

All the delegates and guests were deeply grateful to Mrs. Roy M. Hutchison of Chicago and her committee for the fine hospitality extended to them. No effort had been spared in providing excellent entertainment or in making everyone comfortable.

The officers elected to serve for the coming year are as follows: Mrs. David W. Thomas, Lock Haven, Pa., president; Mrs. Jesse D. Hamer, Phoenix, Ariz., president-elect; Mrs. Eustace A. Allen, Atlanta, Ga., first vice-president; Mrs. J. H. Hornberger, Roebling, N. J., second vice-president; Mrs. A. H. Duemling, Fort Wayne, Ind., third vice-president; Mrs. David Berg, Helena, Mont., fourth vice-president; Mrs. A. Herod, Shreveport, La., constitutional secretary; Mrs. Harold F. Wahlquist, Minneapolis, Minn., treasurer; Mrs. James P. Simonds, Chicago, Ill., director; Mrs. W .W. King, Denver, Colo., director; Mrs. Roscoe E. Mosiman, Seattle, Wash., director; Mrs. J. L. Stevens, Mansfield, Ohio, director; Mrs. Luther H. Kice, Garden City, Long Island, New York, director; Mrs. Eben J. Carey, Wauwatosa, Wis., director.

Respectfully submitted,

CLARICE H. HAMER
(Mrs. Jesse D.)

Book Reviews

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY

(Twentieth Edition)

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY: By W. A. Newman Dorland. A.M., M.D., F.A.C.S., Lieut-Colonel, M.R.C., U. S. Army; Member of the Committee on Nomenclature and Classifacition of Diseaseh of the American Medical Association; Editor of "American Pocket Medical Dictionary". With the Collaboration of E. C. L. Miller, M.D., Medical College of Virginia. Twentieth Edition, Revised and Enlarged. 1668 pages with 885 illustrations, including 240 portraits. Flexible and Stiff Binding. Philadelphia and London: W. B. Saunders Company, 1944. Plain $7.00. Thumb-Indexed $7.50.

The Twentieth Edition of The American Illustrated Medical Dictionary is off the press. The author tells us that its revisions involved additions and alterations on every page. Compared to older editions much has been added. The 240 portraits of men whose names have become linked with the nomenclature of Medicine is a distinctive feature. Every field of medicine and surgery has been covered, and new drugs, and new diseases which have been added to our daily life, as a result of the participation of our medical men in all parts of the world in the present war, are included.

Another important feature is that under each drug are given its source, composition, properties, uses and dose. A complete dictionary could easily be the busy physicians first reference book for information on the spur of the moment.

F. J. M.

MEDICAL DIAGNOSIS

MEDICAL DIAGNOSIS: By Roscoe L. Pullen, A.B., M.D., Instructor in Medicine, Tulane University of Louisiana School of Medicine: Assistant Clinical Director, Charity Hospital of Louisiana at New Orleans; formerly Fellow in Clinical Endocrinology, Duke University School of Medicine and Duke Hospital, Durham, North Carolina. With a Foreword by John H. Musser, B.S., M.D., F.A.C.P., Professor of Medicine, Tulane University of Louisiana School of Medicine: Senior Visiting Physician, Charity Hospital of Louisiana at New Orleans. 1106 pages with 584 illustrations and 12 colored plates. Philadelphia and London: W. B. Saunders Company, 1944. Price $10.00.

This book of over a thousand pages on Medical Diagnosis, and applied Physical Diagnosis, is divided into 30 chapters. It comprises every speciality in the human body. Each chapter is written by a Specialist in his particular branch of medicine. These 30 authors are mostly young, fairly well-known men, many of whom hold teaching positions throughout the country. They not only explain how to make physical examinations, but they describe and interpret the pathological conditions that are to be encountered on examination. The diagrams, illustrations, and colored plates are excellent. This seems to be a book primarily for the student, but for the general practitioner, it is a reference book containing details and information presented in such concise style, that it should be of practical use on any medical man's book shelf. No book has ever been published which approaches the subjects of diagnosis and pathology as this does. Dr. Pullen and his contributors are to be commended on their contributions.

F. J. M.

HANDBOOK ON NUTRITION: A symposium prepared under the auspices of the Council on Foods and Nutrition of the American Medical Association. American Medical Association. Chicago. 1943. 586 pp. $2.50.

Twenty-eight authors have contributed to this symposium on nutrition to bring the knowledge in this field to an up-to-the-minute level for the benefit of students of nutrition and members of the medical profession for whose benefit the text was desgined. The articles first appeared from time to time in the Journal of the American Medical Association.

Chapters on Proteins in Nutrition (Howard B. Lewis), Calories in Medical Practice (DuBois and Chambers), Water and Salt Requirements in Health and Disease (John H. Talbot), The Feeding of Healthy Infants and Children (Philip C. Jeans), Feeding the Aged (Edward L. Tuohy), Principles of Diet in the Treatment of Disease (Thos. D. Spies), and Improving the Quality of Cheap Staple Foods (George W. Cowgill) are among the 25 timely chapters the busy physician will find helpful in his desire to brush up on the latest findings in the field of nutrition as each contributor is a specialist in his field of discussion. Each chapter is comparatively brief, and the type plain and readable in spite of the number of pages. Of additional merit is the fact that each chapter includes a considerable bibliography so the student or physician may pursue his study or investigation further if he wishes. A table on Current Standards for Enrichment and Fortification of Foods is included. All in all, it is a handbook to be highly recommended. The price of the book is modest when both the format and content are considered.

K. C.

MINOR SURGERY: *Edited by Humphry Rolleston and Alan Moncrieff. Published by Philosophical Library, New York.

The entire field of minor surgery, including surgical specialties and anesthesia is presented in an orderly and well-planned manner in Minor Surgery.* The book is intended for the practitioner and this small volume should prove its worth as a refresher course for students, practitioners and specialists in surgery. It begins with a consideration of minor wounds. I cannot agree that minor wounds exist as the smallest wound presents a major problem and may result fatally. Also the last chapter on anesthesia and analysis causes a disagreement. The author's statement that with freshly prepared solutions of 10 per cent soluble hexobarbitone (evipan sidium), or 5 per cent sodium thiopentobarbital (pentothal sodium). "It is possible for one practitioner to make the injection and subsequently to perform the manipulation or operation." Both of these barbiturates are powerful and dangerous and should only be administered by an experienced anesthetist who can give all of his attention to the administration of the drug

and the condition of the patient, then only can these drugs be considered fairly safe anesthetic agents.

E. P. P.

FUNCTIONAL DISORDERS OF THE FOOT: Their Diagnosis and Treatment, by Frank D. Dickson, M.D., F.A.D.S., Associate Professor of Clinical Surgery, Medical School, University of Kansas; Orthopedic Surgeon, St. Luke's Kansas City General and Wheatley Hospitals; and Rex L. Diveley, A.B., M.D., F.A.C.S., Colonel, Medical Corps, Army of the United States; Orthopedic Consultant, European Theater of Operations; Orthopedic Surgeon, St. Luke's, Kansas City General, Research and Wheatley Hospitals, Second Edition, 202 illustrations. J. B. Lippincott Co., E. Washington Square, Philadelphia, London, Montreal. 1944, $5.00.

This is an excellent book which is of particular interest to the orthopedist but should also be of value to the general surgeon or general practitioner who treats disorders of the foot. The book is lucid and arranged in such a manner as to make reference to special conditions readily accessible.

This edition has made some valuable additions to a previously well written and sound treatise on the foot. The addition of chapters dealing with functional disorders of the foot in relation to military service and the disorders in relation to industry are entirely new. In the former, the foot is classified into acceptable conditions and conditions which warrant rejection for military duty. This will be of value to those who are serving on Selective Service Examining Boards and likewise, the chapter on foot disorders to industry will be interesting to the industrial surgeon.

This volume can be highly recommended to those interested in the examination and treatment of the foot.

J. L-S.

THE LOIS GRUNOW MEMORIAL LIBRARY
NEW BOOKS

BACKACHE AND SCIATIC NEURITIS, by Philip Lewin. 1943.
That complex mechanism—the back—is of equal interest to the orthopedic surgeon, gynecologist, urologist, obstetrician, neurologist, and roentgenologist. Here it is in all its complexities and by an authority!

RECONSTRUCTIVE SURGERY OF THE EYELIDS, by W. L. Hughes. 1943.
The title is self-explanatory—the subject is timely.

ORTHOPEDIC OPERATIONS, by A. Steindler, 1940.
The purpose of this book is to supply clear

and exact information on how, when, and with what results a given operation should be performed in a given situation. The author fulfills this purpose.

ACUTE INFECTIONS OF THE MEDIASTINUM, by H. Neuhof and E. E. Jemerin. 1943.
Mediastinal infections, so often regarded as strange lesions situated in a more or less inaccessible part of the body and characterized by high mortality, are approached and studied in a rational manner. The authors have charted a practical course that can be easily followed by any doctor encountering or suspecting mediastinitis.

CLINICAL NEUROLOGY, by I. S. Wechsler. Fifth Edition. 1943.
"The brief period of four years since the last edition appeared witnessed a number of advances in neurology: Chemotherapy of meningitis is practically new; headache is better understood; and electroencephalography, 'degenerative' diseases, and the automatic nervous system, all have received further study."

ORAL AND FACIAL DISEASES AND MALFORMATIONS, by G. V. I. Brown. Fourth Edition. 1938.
Plastic reconstructive surgery hac come to be an important part of surgical practice. The general practitioner must be informed as to the possibilities of improving or correcting disfiguring scars, burn contractions, etc. Here is a text which offers a safe guide to the diagnosis and treatment of such conditions. Profusely illustrated.

APPLIED ANATOMY OF THE HEAD AND NECK—for practitioners of dentistry, by H. H. Shapiro. 1943.
This text is specifically designed to relate the anatomy of the head and neck to the various fields of dentistry. The anatomic detail selected for description has been confined to those features which are of the most significance in diagnosis and treatment.

INTERNAL MEDICINE IN DENTAL PRACTICE, by B. I. Comroe, L. H. Collins and M. P. Crane. Second Edition. 1943.
So often it is the dentist who first has the opportunity to observe manifestations of systemic disease! The authors feel that for this reason there should be closer cooperation and understanding between the internist and dentist. And so—a book—which points out the oral manifestations to the dentist in a clear, concise manner.

CLINICAL PARASITOLOGY, by C. F. Craig and E. C. Faust. Third Edition. 1943.
Here they are—those parasites which provide such clinical and public health problems! The symptoms caused by their presence and the important methods of diagno-

sis, treatment and control are clearly described.

GERIATRIC MEDICINE; diagnosis and management of disease in the aging . . . . edited by E. J. Stieglitz. 1943.

The authors feel that during the next fifty years physicians will be called upon to treat relatively larger numbers of cases of cardiovascular renal diseases, cancer, diabetes, serious injuries that result from falls of the aged, and of senile psychoses; but relatively fewer cases of tuberculosis, syphilis, typhoid fever, malaria, other acute infectious processes, and of diseases due to dietary insufficiencies. The medical profession should prepare itself for this important change in the pattern of its activities. Here is the text with which to begin such a preparation.

COLLEGE OF SURGEONS

The Ninth Annual Assembly of the International College of Surgeons will be held on October 3, 4, 5, 1944 at the Benjamin Franklin Hotel in Philadelphia, Pa. The program will be devoted to War, Rehabilitation and Civilian Surgery.

This Assembly, sponsored by the United States Chapter of which Thomas A. Shallow, M.D., F.A.C.S., F.I.C.S., of Philadelphia is President, has set up its Arrangement Committee with Dr. Rudolph Jaeger as General Chairman. Dr. Jaeger will be inducted as the incoming President of the United States Chapter at the Convocation on Wednesday evening, October 4. The new president came to the Jefferson Medical College from Denver, Colo., where he specialized in Neurosurgery.

Eminent surgeons in Government, Military and Civilian practice have been invited to attend and present papers pertinent to surgery in their particular field of endeavor.

The Chairmen of the various committees are: Dr. William Bates, President-elect of the Pennsylvania Medical Society, is chairman of the Program Committee; Dr. John Royal Moore, Philadelphia, Exhibits; Dr. Moses Behrend, Philadelphia, Hospital Clinics; Dr. William L. Martin, Philadelphia, Registration; Dr. Ernest F. Purcell, Trenton, N. J., Convocation; Dr. John E. Loftus, Philadelphia, Entertainment; Dr. Harold D. Corbusier, Planifield, N. J., Rehabilitation; Dr. Benjamin Shuster, Philadelphia, Housing; Dr. William F. Whelan, Motion Pictures. The medical profession is invited to attend the Assembly and its sessions.

TANTALUM AVAILABLE FOR CIVILIANS

Tantalum plates, foil, screws and wire to repair broken bones, nerves and skulls will shortly be available to civilian surgeons through a recent allocation of the War Production Board, according to an announcement made by Dr. Gustav S. Mathey, President of the Johnson & Johnson Research Foundation, New Brunswick, New Jersey.

The Johnson & Johnson Research Foundation is a non-profit organization, founded in 1940 to endow research in universities and hospitals and to disseminate summaries of findings to members of the medical profession. Dr. Mathey states that by an agreement between the Ethicon Suture Laboratories, Johnson & Johnson subsidiary, and the Fansteel Metallurgical Corporation of North Chicago, the availability of tantalum for civilian surgeons is assured at an early date.

Tantalum has assisted surgeons to return to active life many cases which in the last war would have been disfigured and incapacitated for life. Lost portions of the skull, ears, noses and other parts of the face are being replaced with tantalum. One veteran has a tantalum "belly wall". Nerves which control motion in arms and legs are stitched with tantalum thread and protected while healing with tantalum cuffs. Facial paralysis is relieved by small, saddle-shaped pieces of tantalum and wire used to pull the corners of the mouth to a normal position. This stops the unpleasant drooling and facial distortion which go with the condition. Cleft palates also are being corrected.